ACUTE ANTERIOR POLIOMYELITIS.

A Review of our Present Knowledge with Illustrative Cases

Thesis presented for the Degree of Doctor of Medicine,

University of Edinburgh.

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INTRODUCTION.

There are few diseases of early life that present so many interesting problems as does Anterior Poliomyelitis, and there are still fewer in which our knowledge as to causation and prevention has made so little advance in spite of the many and thorough investigations which have been made into the spontaneous disease in the human subject.

It was not until comparatively recently that the association was noted between the obscure early symptoms of this disease, and the flagrant resultant paralysis. Poliomyelitis, seven years ago, was not definitely classed as an infectious disease: and it is only within the last few years that extensive research has been made into the pathology and cause of this important scourge to infant life.

Unfortunately, there is little in the early febrile stage to distinguish this, from the many other feverish attacks so common during childhood, and it is only when the devastating paralysis supervenes that the true nature of the complaint is made evident. As a result of this, the disease - except in epidemic form, when the profession is exceptionally alert to the possibility of infection - is rarely diagnosed in the early stages; and therefore the practitioner's efforts are mainly palliative.

The child is crippled, and in order that everything may be done to prevent or lessen deformity, the case is transferred from the physician to the orthopaedic surgeon. In consequence, the medical aspect of such cases has been apt to be overlooked. Within the last few years, however, progress has been rapid; epidemics have been prevalent, and skilled observers have studied every aspect of the affliction. Foremost in the field we find Wickman, Müller and Flexner, to whom we are indebted for much of our present knowledge. The disease has been produced experimentally in monkeys, and it has been found that in these animals, the clinical results and pathological findings correspond with great accuracy to those found in man. Attempts to transmit poliomyelitis to other animals have been unsuccessful.

The preliminary fever has been conclusively proved to be the first expression of the disease, and should an effective treatment be discovered in the future, it is to this pre-paralytic stage that our efforts must be directed. We at present possess no such remedy.

As we have just seen it is of great importance to the patient that we diagnose his case early, but an early recognition of poliomyelitis must be encouraged for another reason.

It has been proved beyond doubt that the disease is of an infective nature, and consequently any attempt at isolation should be commenced in this stage: if this quarantine be rigorously carried out, much distress and suffering may be averted.

There is also a certain class of case which causes much confusion, and is of great danger to the community at large. This is the abortive type of the disease, when the patient never develops paralysis at all, but is just as contagious to his neighbours as if he did so.

We are unable to state in what proportion these abortive cases occur, for they are rarely diagnosed except in epidemics and then only with absolute certainty if the neutralization test be resorted to, a proceeding which is not often practicable.

The literature on this fascinating disease is enormous, and in the following pages a brief attempt will be made to review our present knowledge on the subject, and to give special attention to its clinical and experimental aspects. Several cases which have been under the author's care are detailed at length towards the end of this exposition.

One case being of exceptional value, as the disease proved fatal and at the post mortem the cord was removed and sections of this were cut. Microphotographs of the most interesting of these are reproduced.

Another case of some rarity is that of a man, aged 22 years, proving that the disease is by no means limited to the very young, as was formerly believed.

NOMENCLATURE.

This affection has at various times had many names, and perhaps a list of the most important would be of interest. These include:-

1. Essential paralysis of children. Rileict and Barthez, 1853.
2. Infantile spinal paralysis. Heine, 1860.
3. Myogenic paralysis. Bouchert, 1862.
4. Anterior spinal paralysis. Cornil, 1863.
5. Atrophic fatty paralysis of children. Duchenne, 1864.
6. Acute anterior poliomyelitis. Schultze, 1876.
7. Polioencephalomeningo myelitis. Cadwalder, 1908.
8. Epidemic paralysis. Ball, 1909.
9. Acute epidemic paralysis. Krause, 1909.

The names that survive today and are in common use, are Heine's - Infantile Spinal Paralysis, and Schultze's Acute Anterior Poliomyelitis.

HISTORICAL.

Poliomyelitis has been gradually separated in the past 70 years from a large number of palsies, long intervals of time having elapsed between the discoveries.

1840
Heine[1] of Darmstadt, Germany, wrote an article dealing mainly with the orthopedic aspect, but giving a good account of the acute stage. He suggested a serous exudation into the cord as being the cause.

1843
Colmer[2] described an epidemic, but except for this there is practically no literature on the disease until -

1870
When Charcot[3] working at the pathology, stated that there was a primary degeneration of the anterior horn cells of the cord.

1884.
Strümpell[4] compared anterior poliomyelitis with acute encephalitis, and blamed a common causal agent: he suggested an external source of infection.

1890.
Medin[5] published a good clinical account of the disease and described the spastic types.

Accounts of small epidemics in France, Italy, Germany, and the United States were frequent for the next few years, but the next important contribution was in -

1905
by Wichman[6]. Studying many Swedish epidemics, he described fully the epidemiology and pathology and drew attention to the abortive cases. He also demonstrated the contagious nature of this disease.

Then in

1909
Landsteiner and Popper[7], Flexner and Lewis[8], and Strauss[9] described the experimental production of the disease in monkeys.

Earlier records of epidemics of this disease were given us by Medin[10] and Brieglib in Europe, and by Colmer[11] and Calverley[12] in America.

Wickman[13] in a convincing monograph in 1907 proves the contagious element.

In the long series of important epidemics perhaps the following five stand out most conspicuously[14].

Wickman's Swedish	Epidemic	1905
Lorett's Massachusetts	"	.....1907-8-9.
Kraus's Westphalian	"	1908
Lindner and Mally's Steys	-	1908
Potpeschnigg's Steiermark	-	1909

Each has added a little to our store of knowledge and removed some fallacy from the path which is beset with many difficulties.

In the November of 1909 three observers in different cities, working independently, succeeded in transferring the disease from one monkey to another.

1. Flexner and Lewis[15] in New York.
2. Leiner and Von Weisner[16] in Vienna.
3. Landsteiner and Levaditi[17] in Paris.

It is therefore to the last five or seven years that we owe so much of our knowledge, and although many useful discoveries have been made there is still much that defies our present methods of research.

CLASSIFICATION.

There are several forms of clinical classification of cases of anterior poliomyelitis, one of the most commonly accepted being that of Wickman - eight types. He bases his method partly on the anatomical findings and partly on the clinical symptomatology, i.e. (1)

(1) Spinal poliomyelitic form.

(2) Ascending form similar to Landry's paralysis.

(3) Bulbar and pontine form.

(4) Encephalitic form.

(5) Ataxic form.

(6) Neuritic form.

(7) Meningeal form.

(8) Abortive form.

This list seems unnecessarily complicated and a more concise method is that of Müller.

He simplifies matters by bringing cases under four heads -

(1) Spinal.

(2) Bulbar.

(3) Cerebral

according as the symptoms point chiefly to one or other of these places as the site of the lesion, and lastly

(4) Abortive cases

There are however, two main clinical types:-

(1) Those cases involving chiefly the Upper ~~nerves~~ neurons.

(2) " " " " " Lower "

These often overlap and it is no uncommon thing to see spinal lesions complicated by cerebral involvement.

When the lower motor neurone is attacked as is the case in the majority of patients, a flaccid paralysis occurs, but in the more rare instances where there is only a cerebral lesion a spastic form of paralysis is the result: here in a few cases the focus may be in the cortex but there is reason to believe that the pyramidal tracts are usually the injured areas.

Spastic and flaccid paralyses have been noticed in the same epidemic, and on several occasions have been found in the same patient.

The exceedingly dangerous and often missed abortive cases must also never be forgotten - whatever classification be adopted, as these are of the utmost importance.

TRANSMISSION.

The ways and means by which the virus of poliomyelitis can be conveyed from one subject to another, are still sub judice. In epidemics which have been exhaustively investigated by eminent observers, various solutions have been put forward.

Infection may be direct or indirect. The work of Flexner and Clark[18], and of Kling, Wernstedt and Pettersson[19] seems to prove conclusively that the chief source of entry and exit of the virus to and from the central nervous system is by the naso-pharyngeal mucous membrane, for they have repeatedly demonstrated the presence of the virus in this mucosa, in the secretion from the cavity of the nose and pharynx and in the pharyngeal tonsils. They have shewn that the experimental disease in monkeys can be readily produced by swabbing the nasal mucosa with the virus.

Landsteiner, Levaditi and Pastia[20] have also demonstrated the virus in the tonsils of fatal cases of this disease.

A short summary of the experimental work connected with the transmission of poliomyelitis to lower animals is of great interest.

In May 1909, Landsteiner and Popper[21] published two successful inoculations of monkeys with spinal cord from a child dead of the disease. They injected the

emulsified cord into the peritoneal cavity, with the result that the monkeys became paralysed, and after death lesions of the cord were discovered. They however failed to transmit to another generation. Previously to this very little work had been done in this direction, and when such had been attempted, failure had been the result; e.g. in 1907 Flexner and Lewis tried to obtain the disease by injection of the cerebro spinal fluid obtained by lumbar puncture, but their results were negative.

In September 1909 Flexner(22) however, secured the spinal cords from two human cases that had died from the disease. An emulsion in salt solution was made from them and it was inoculated into monkeys. This inoculation was performed through a trephine opening into the brain. The results were highly successful - paralysis followed and characteristic changes were demonstrated in the central nervous system.

To carry the experiments further the cords of the paralysed monkeys were taken, emulsified as before and inoculated into other monkeys with equal success. It has been possible to transmit the disease for many generations of monkeys (twenty-five or more) in this manner. The cortex of the brain is also active when injected.

M'Intosh and Turnbull(23) were the first in Great Britain to record successful inoculation of the virus

into monkeys. They obtained the emulsified cords from English cases and succeeded in transmitting the disease through three generations of monkeys.

Further experiment shews that it is not necessary to inject the virus into the brain or cord direct, the effect is the same if inoculation takes place into the peritoneal cavity, into vessels or around nerve trunks. It is probable that other avenues - skin, respiratory and digestive tract may allow of entry, but this still remains to be proved. An interesting fact, however, is that in whatever site inoculation occurs, vessel, nerve, etc. the characteristic lesions are always found in the cord and medulla, causing the typical symptoms and paralyses.

Osgood and Lucas[24] confirmed Flexner's view that the naso pharyngeal mucous membrane was the chief channel of infection from without, and they managed to transmit the disease by means of a naso-pharyngeal filtrate from monkeys who had recovered from acute stages of acute anterior poliomyelitis, in one case for 6 weeks, and in another for 5½ months, thereby shewing that the virus can remain in an active form in the naso pharyngeal mucosa for some considerable time.

A practical point however, is that although the virus does remain, its virulence rapidly decreases and to all intents and purposes one month's isolation is usually sufficient. (Trethowan)[25].

Rosenau[26] attempted to transmit the disease to monkeys by inoculation with the naso pharyngeal and buccal secretions of 18 human cases. His method was as follows:-

Several washings were taken from the above cavities with normal saline. The floor of the nose and mouth was swabbed with sterile dabs and the secretion obtained added to the washings. The material thus collected was cooled, packed in ice and taken to the laboratory, then passed through a Berkfelt filter warmed to 30°C. and injected into the brain and peritoneal cavity of seven monkeys (Macacus Rhesus).

The result was extremely disappointing, in not one single case did any symptoms of poliomyelitis appear. It must, however, be remembered that the virus was in an extremely dilute form and this may have caused the failure.

Levaditi[27] following out the same line of research found the pathogenic agents contained in the nasal mucous membrane, but not in the nasal secretion. He suggested permanganate of potash as a prophylactic.

The bulk of this evidence, in spite of Rosenau's failure, goes to shew that the naso-pharyngeal mucous membrane is a source of danger, and is probably the chief means by which the virus reaches and leaves the central nervous system.

The way in which the virus accomplishes this journey is probably by the lymphatic channels surrounding the nerves of smell that pass from the olfactory lobes to the nasal mucous membrane.

Flexner[28] proved that this connection exists by his experiments with the Diplococcus Intracellularis. In these experiments monkeys were injected intracerebrally. Subsequently the Diplococcus was found in the leucocytes and also free in the naso pharynx of the victim.

The method of spread in the brain is mere conjecture, but probably the cerebro spinal fluid acts as the vehicle.

It has also been proved that the reverse route is traversed by the virus, for in monkeys infected intercerebrally the nasal mucosa is highly virulent. Probably it is from this source that the causal agent spreads, it infects the oral secretions, is swallowed, and so intestinal inoculation occurs and the mesenteric lymph nodes are reached. Here the virus is often found, a notable exception to other internal organs. (Flexner and Lewis).

It has been demonstrated repeatedly that the keeping of healthy and infected monkeys together does not lead to infection of the healthy .

Repeated attempts have been made to implant the virus on other animals, but so far without success. Krause and Meinicke[29] and Lentz and Huntemüller[30] claimed that rabbits may be infected with the virus, to the effects of which they may succumb.

On the other hand Landsteiner and Levaditi[31] and Römer and Joseph[32] failed completely to produce the disease in rabbits by inoculation of tissues of the spinal cord and brain from cases of experimental (monkey) poliomyelitis.

Marks[33] made some exhaustive experiments in order to try and clear up this contradictory evidence, and he found that in no case did he get any paralysis in the rabbits, or any localised nerve lesions after death. The animals that died usually did so on the 8th and 15th day, death being preceded by a convulsive seizure. Post-mortem there was hyperaemia of the cerebral cortex but nothing else.

He discovered, however, that these rabbits can carry the disease, even if they do not succumb to it, and he succeeded in transmitting the virus through a series of rabbits - six in number, and then transferring it back to a monkey, the effect being a typical paralysis with characteristic nerve lesions.

He suggests that as these experiments prove that the virus of the disease can survive in a domestic

animal without that animal shewing any symptoms, it is conceivable that there is some animal in relation to man that may act as a reservoir for the virus, which only occasionally and under special and peculiar circumstances transmits it to human beings. That such an animal reservoirs of pathogenic organs do exist has been proved by the establishment of the connection between infected goats and Malta fever(34).

This suggestion is vague but extremely interesting and opens up a new line of thought in the attempt to fathom the transmission of the disease.

Passive Human Carriage of Virus.

The manner in which the virus of poliomyelitis is conveyed to the infected persons has not been fully worked out, but as the presence of this virus has been established upon the mucous membrane of the nose, throat and intestines of persons ill of poliomyelitis, and also in sweepings of rooms in which cases had been recently kept, (Neustaedter and Theo(35)) it is reasonable to suppose that the virus is capable of leaving the body in a manner that permits of its persistence as dust.

The discharges of the affected persons contaminate their surroundings; and when dried can retain the active virus for a considerable time.

Not only can the infection be transmitted from

infected persons, but Flexner and Clark[36] have demonstrated experimentally that the naso-pharyngeal mucous membrane of healthy contacts can retain the virus in an active form and so be a source of danger to those who are more susceptible to this disease.

In their experiments they made washings of the naso-pharyngeal mucous membrane from the mother and father of a child suffering from the disease, and after filtering through a Berkefeld filter they infected 1.5 c.c. of this saline solution into the sheath of each sciatic nerve, and 140 c.c. into the peritoneal cavity of a monkey (Macacus cynomolgus). Ten days later the animal developed a typical palsy and at the autopsy the central nervous system shewed characteristic changes.

The emulsion of the cord and medulla of this monkey was injected in the same way into another animal with similar result to the first.

These experiments prove conclusively what has been long suspected, that passive human carriers of the disease do exist.

Kling Pettersson and Wernstedt[37] have carried out similar experiments with like results.

They have also observed that the virus occurs with great constancy in the washings of mucus from the large and small intestine, and it is presumable that it reaches these sites by means of the swallowed secretions of the

mouth and nose. The virus survives the action of both the gastric and intestinal secretions and persists for a time in these organs. In human beings it leaves the body in the excreta and is therefore a source of infection, unless these be disinfected and destroyed.

Insect Transmission.

The question of Insect Transmission of the disease is very important and has been exhaustively worked at by Howard and Clark(38).

They conducted a series of experiments with domestic flies, mosquitos, lice and bedbugs.

These are of great value because in studying this question of transmission we are confronted with a complex problem, for not only do we find cases which can be traced through direct or indirect contact with persons suffering from the disease, but we also meet with sporadic cases occurring over widely scattered areas and apparently well isolated from any acute source of infection.

Should insect transmission be proved possible, this sporadic occurrence may be easily explained.

A short resumé of these observers' results is of great interest.

House flies. (Musca domestica).

These were allowed to feed upon infected cord: later they were killed, ground up with sand and normal saline and passed through a Berkefeld fliter. The resultant liquid was injected into the brain of several monkeys. These developed typical palsy.

In two cases only, the flies' viscera, removed with aseptic precaution, were used with like result, showing that the virus can remain alive in the body of the fly for at least several hours after ingestion.

Mosquitos. (Culex Pipiens and Culex sollicitans).

Here the process was very much the same. The mosquitos were allowed to feed, then ground up, filtered and injected. Fifteen monkeys were used and not one of them shewed any symptoms of poliomyelitis.

Lice. (Pediculus vestimenti and Pediculus capitis).

Here the insects were allowed to feed on infected monkeys for several days, then killed, ground up in salt solution, filtered, and injected intracerebrally. The results were absolutely negative.

Bedbugs. (Cimex lectularius).

Same method employed as with lice. Sixteen monkeys injected and only one shewed evidence of poliomyelitis. This one became paralysed and shewed characteristic cord changes at the autopsy. A glycerinated

suspension of the cord of this monkey was taken and injected into another Macacus Rhesus, causing definite paralysis and central nervous system changes.

The deductions from these experiments are helpful, for we have seen that the domestic fly can carry the virus of poliomyelitis in an active state for several days upon the surface of its body, and for several hours within its gastro-intestinal tract; it is therefore only reasonable to suppose that this transmission can occur in nature.

The experiments with the mosquito and lice were negative and inconclusive, but with the bedbug, although only in one case did infection occur, yet this proves that a blood-sucking insect can obtain the living virus from the blood, and retain it for several days in an active state.

Frost[39] performed some similar experiments with the stable fly (Stomoxys calcitrans). He allowed the flies to bite infected monkeys and then placed them in a cage with healthy animals. Three of these latter developed typical symptoms, seven, eight and nine days respectively from the date of first exposure, and emulsion of their spinal cords transmitted the disease to other monkeys.

In the treatment of an epidemic of poliomyelitis

we must, therefore, in order to leave no source of infection, treat these flies and lice with extreme suspicion and where possible attempt to eradicate them altogether from the surroundings of the patients.

VIRUS.

Causal Agent.

The causal organism in epidemic poliomyelitis is undoubtedly ultramicroscopic,-if it exist at all,- for in spite of prolonged and diligent research on the part of many distinguished observers, the microscope has failed to reveal any such agent.

Flexner[40] adopted another line of investigation. It is known that the viruses of rabies and vaccinia (in neither of which any organism is visible) withstand the action of glycerine very well, whereas on the other hand bacteria withstand it badly. He therefore tried the effect of glycerine on this unknown quantity.

He suspended the comminuted cords of monkeys suffering from poliomyelitis in glycerine, leaving them there for seven days. The glycerine was then washed away with salt solution, the suspended matter centrifugalised and so recovered.

To prove that the virus had suffered no hurt in the process the cord subjected to the above glycerination was injected into a monkey and this animal developed a typical paralysis.

As further proof that the causal agent is probably not of bacterial origin, the same observer decided to try the effect of filtration through the Chamberland and Berkefeld filters. These filters prevent the passage of all known bacteria, and so if the filtrate was proved to be still virulent and capable of producing the disease then it would be unlikely than any organism was responsible.

The cord of a monkey dying from acute anterior poliomyelitis was triturated with sterile quartz sand mixed with salt solution; it was well shaken and then passed through a Berkefeld filter. This bacterium free filtrate was injected into the brain of a monkey and characteristic lesion occured on the ninth day. Therefore the causal agent in epidemic poliomyelitis probably belongs to that group known as "filterable viruses".

It is believed that eighteen diseases are due to these filterable viruses. There are only at present three known in man, i.e. (1) dengue fever, (2) yellow fever and (3) poliomyelitis, the remaining fifteen occur in lower anmials, e.g. foot and mouth disease, cattle plague, rabies, vaccinia and chicken plague.

They are extremely resistant. They stand drying well and are only killed with difficulty by disinfectants.

The virus of poliomyelitis retains its virulence after freezing at a temperature of -2° -4°C. in a Frigo apparatus for forty days, showing that although the disease is less prevalent in winter, it is not destroyed by cold.

The virus can also still transmit the disease after a lengthy suspension over caustic potash in a dessicator. It resists as we have seen, drying, light and chemical action. It has been proved to exist for months in dust. Diffuse sunlight has no effect upon it. It can withstand pure glycerine and a 0.5% solution of carbolic acid for months, and when animal tissues containing the virus undergo softening and degeneration the virus survives. On the other hand it is destroyed on being exposed to a temperature of 45° - 50°C. for thirty minutes, also by a 1% solution of hydrogen peroxide.

After recovery from this class of infection there is a high immunity to the disease, this being possibly due to a microbial agent acting on and destroying the parasite.

Cultivation of Virus.

It has been impossible to cultivate the virus of poliomyelitis up to quite recently.

Flexner and Noguchi(41) have lately worked patiently

at this and in a note published this year, appear to have succeeded.

It must be remembered that the causal agent belongs to the class of filterable viruses and passed readily through a Berkefeld filter. These observers employed for their experiments, tissues from the brain and spinal cord of human beings dead from the disease, and from monkeys the subjects of the experimental disease. The cords were rendered free from ordinary bacterial contamination by suspension in a 50% glycerine solution for some months.

The culture media used consisted first of sterile, unfiltered ascitic fluid or of brain extract to which fragments of sterile rabbit kidney and a layer of paraffin oil had been added, and of these plus 2% nutrient agar agar in proportion of 1 to 2.

The first media permit of a slow growth not visible to the naked eye, while the second (which are unsuitable for obtaining the initial growth) yield after several days visible minute colonies clouding the tubes. The cultivations are conducted under anaerobic conditions.

The minute colonies are composed of globular bodies 0.15 - 0.3 of a micron in size. They are in a variety of arrangements, single, double, short chains and masses. Monkeys have been inoculated with these cultures.

Cultures from human tissues in the third, and from monkey tissues in the fifth generation, have caused typical experimental poliomyelitis in the monkey. At the autopsy the central nervous system shewed lesions characteristic of the disease, and from the nervous tissue of these animals, other monkeys were successfully inoculated and pure cultures recovered.

It may be that a certain quantity of the original virus sufficient to cause paralysis in the inoculated monkeys, was carried over with the culture of the globoid bodies, and further experiments with later generations of the cultures are now in progress.

PATHOLOGY.

The first investigations in this department were made on chronic cases of the disease, i.e. cases in which the paralysis had been present for some months or years, and the pathological findings were mostly confined to atrophic scars found in the anterior horns of the cord.

The central nervous system lesions were usually the only ones described in a post mortem report on a case of poliomyelitis. Early observers in this field of the work were Harbitz and Scheel[42], Wickman[43], Strauss[44] and Rissler[45]. They planned out the site of the injury to the cord pretty accurately and although noting the presence of lesions in other parts of the body at autopsy, did not attach to these the importance they deserved.

Lesions in Central Nervous System.

The cerebro spinal fluid is not usually increased in amount, the meninges are often slightly congested. On section brain and cord have a rather moist appearance and are slightly oedematous. The gray matter projecting above the white, scattered haemorrhages may often be observed.

The view that infection takes place from the naso-pharyngeal mucous membrane by means of the lymphatics which accompany the olfactory nerve is almost generally

accepted. The anatomical findings corroberate this view, as it is in the meninges that the first changes are found. These are hyperaemia and the collection of a large number of mononuclear cells of small size in the lymph spaces surrounding the blood vessels of the lepto-meninges.

These lymph spaces are anatomical processes of the arachnoid spaces and their contents are in direct communication with the cerebro spinal fluid, so that there is at first an acute interstitial meningitis. The site at which it is most commonly found is the anterior surface of the spinal cord, and more especially in the anterior fissure from which the larger vessels enter the cord. The vessels in the meninges over the posterior and lateral aspects are also sometimes affected but to a lesser degree. The cord receives its blood supply from the meningeal vessels and in consequence the inflammatory change spreading along the vessels enters the cord, so one of the very earliest changes noted on section in the cord itself is perivascular infiltration and hypereamia.

In many instances the cells are so numerous that they press upon the accompanying vessel and obstruct its lumen. The haemorrhages so frequently found may be due to some toxic change in the intimal lining of the vessels and the accompanying oedema may also be explained on the same hypothesis.

So there are three primary changes in the spinal cord due to the action of the virus, - cellular exudate, oedema and haemorrhage, these can all be traced to vascular changes.

It is still a moot point whether the changes - degeneration, etc. of the nerve cells are due to the direct action of the virus or are secondary to the above vascular disturbance.

These intimal changes may lead to thrombosis of the anterior spinal and other main arteries, or an embolus may enter these vessels with sudden result.

The vascular theory is the more likely solution, and if this view be taken it explains the frequency with which the cervical and lumbar segments of the cord are the sites of injury, for here the blood supply is more generous than in any other part.

The irregular distribution of the vessels supplying the cord at different levels may explain the frequency of the asymmetrical paralyses. Granted that the vascular disturbance be the primary factor the nerve cells degenerate for two reasons, (1) pressure from the exudate and oedema, (2) anaemia from constriction of their blood supply. The resulting paralyses depend for their severity on the amount and length of time that the pressure on the cells lasts. If the exudate be absorbed quickly the cells may recover.

If there is, however, prolonged pressure and anaemia then complete cellular necrosis occurs. All stages of degeneration are seen under the microscope.

The motor cells degenerate, the nucleus undergoes chromatolysis, and the cytoplasm becomes granular and vacuolated. In a later stage the affected cells are completely disintegrated, phagocytes remove the detritus. The non medullated nerve fibres are swollen and the neuroglia increases. The affected horn in old standing cases becomes smaller than that of the opposite side. Nerve cells and fibres are absent, vessels are thickened, anterior roots of the spinal nerves are atrophied, and there is a descending degeneration of motor fibres.

There is no hard and fast circumscribed area in which these changes occur, but they are more or less diffuse in both gray and white matter.

Similar changes are seen in the medulla pons and brain. Lesions here, however, causing paralysis are rare.

In the great majority of cases there are also marked pathological changes in the posterior root ganglia; they are very similar to the cord changes.

There is a perivascular round celled infiltration of the ganglie from the meninges, there is the exudation and the cell necrosis, and disintegration, and the neurophagic polymorphs are present.

Pain.

It has been suggested that the pain in the acute stage of the disease may be due to the involvement of these sensory ganglia. Another cause of the pain may be the cellular infiltration found along the nerve roots.

Changes in other Organs.

These are most frequent in the lymphoid tissue scattered over the body. Peyers patches of the intestinal mesenteric glands, bronchial, cervical, axillary and inguinal lymph glands often shew acute swelling.

The spleen is often enlarged showing on section raised translucent Malpighian corpuscles.

The thymus is no exception to the general lymphoid change.

Microscopically in the lymph tissue there is disintegration of cells in large numbers, and in many of the necrosing areas there is an invasion of polymorphonuclear leucocytes.

Cloudy swelling is common in the parenchymatous organs and especially in the liver, here also necrotic areas have been described, these being closely dependent on the vascular supply. The lesion may consist of only one or two disintegrating cells or a great part of lobule may be affected.

It is practically certain that these changes are due to the virus of poliomyelitis, for they have been found again and again in man and also in the organs of monkeys infected experimentally with the disease. (Flexner[46]).

We must therefore regard the disease as a generalised infection of the whole system and not as a nervous entity.

Flexner and Clark[47] describe the pathological findings in a dog which developed clinical symptoms closely simulating poliomyelitis. The autopsy shewed changes in the spinal cord and intervertebral ganglia, resembling but not identical with the lesions of poliomyelitis in man.

These consisted of (1) perivascular infiltration, (2) haemorrhage, (3) oedema, infiltration and necrosis of ground substance followed by invasion of large phagocytic cells, (4) necrosis of ganglion cells.
Inoculation of the affected spinal cord into the nervous system of puppies and monkeys was not followed by the production of paralysis or obvious symptoms of the disease.

So on the whole the conclusion is that it is extremely doubtful whether this was a true poliomyelitis.

IMMUNITY.

Numerous experiments have been conducted to determine the kind and degree of immunity which is produced by the inoculation of the virus.

A large number of monkeys have been employed for this purpose and re-inoculations have been made at periods varying between two days and five months, after the first appearance of the paralysis. In no instance have fresh symptoms been noticed or any increase in the paralysis, which suggested a renewal of the attack. It is an undoubted fact that immunity to a second attack is present even in those cases of poliomyelitis where there is no paralysis.

The immunity depends upon the presence in the blood of certain protective agencies which are capable of neutralising the virus.

This has been demonstrated by mixing an active filtrate containing the virus with the blood serum either of children or monkeys who had recently recovered from the disease. The mixture was incubated for an hour at 37°C. and then injected into the brains of normal monkeys, and it was found that in no instance did paralysis follow.

Further, Netter and Levaditi[48] have shewn that the blood serum of a child after an abortive attack of poliomyelitis also contains a similar protective agency.

Active immunization has been performed by Flexner[49] in several instances. The virus was injected subcutaneously in gradually increasing doses for several months and after this a full intercerebral injection of the filtrate failed to cause paralysis. As we have seen monkeys recovering from the paralysis are imsusceptible to re-inoculation with the virus, i.e. they have developed an artificial immunity. The blood serum of children who have had the disease can convey immunity to the monkey even when large doses of virus are injected.

Flexner's conclusions were as follows:-

(1) If the quantity of virus injected into the brain twenty four hours before serum treatment be begun, is not in excess of a given dose - the action of the virus can be prevented.

(2) Infection of the meninges from the nasal mucosa can be prevented by serum injection.

(3) Normal monkey serum has no such effect.

(4) Horse serum is entirely without preventitive action and tends to hasten the onset of paralysis.

These observations suggest the possibility of producing a therapeutic serum by inducing an active immunity in some of the lower animals. Experiments upon the horse, rabbits and chickens have shewn that these animals do not yield such principles, but on the other hand there are indications that the sheep may react more favourably. It has been found that normal sheep's serum possesses a definite neutralizing power when mixed

with filtered virus, and that this power can be reinforced by injection of emulsion of the spinal cord of recently paralysed monkeys, so that it only remains to be determined whether this augmentation can be carried out to such a degree as to produce a serum applicable to the spontaneous disease in human beings.

These immunity principles are probably in the nature of antibodies. Poliomyelitis is therefore possibly a self limiting process due to the elaboration in the blood of these antibodies.

They exist for many years in the blood. It has been stated that they are always found in the cerebro spinal fluid but this is unlikely, they are certainly found at times in the cerebro spinal fluid but chiefly in the acute stages, and therefore due to the increased permeability of the vessels of the central nervous system.

Neutralisation Tests.

We have hereby an excellent test of whether or no a case is one of acute anterior poliomyelitis.

The test is performed by mixing the blood serum of the suspected case with the filtered virus, incubating at 37.6 for a few hours and injecting into the monkey's brain.

If the virus be neutralised the case is one of anterior poliomyelitis, if the virus be not neutralised

and paresis follows, then the suspected case is probably not anterior poliomyelitis.

This rule is not infallible but it is useful and of help, especially in the detection of abortive cases, for here there is little else to guide one and unless these cases be diagnosed early and prevented from spreading the disease broadcast, much harm may be done.

It is not always possible to do the neutralisation test but where it is practicable the suspect should certainly be subjected to it.

PRODROMATA OF THE ATTACK.

The incubation period in man is hard to gauge. Wickman believes it to be from 14 days. Müller from 5 to 10, and Flexner in his experimental inoculations gives an average of 8 - 9 days with occasional variation of from 3 - 30 days depending on the toxicity of the virus. The average is probably about one week.

This period is usually followed by very definite prodromal symptoms often only elicited by careful questioning of the parents, for they are in many instances so slight as to be overlooked.

In a few cases there seems to be no such stage, and the paralysis is the first thing noticed. The recognition of such a period is of the utmost importance because in the first place measures may be taken to isolate suspected cases, and secondly, the appropriate treatment commenced, in order to try to control the disease before an extensive destruction of nerve cells takes place.

The average length of time during which these preliminary manifestations are present is from 1-3 days. They may increase in severity until paralysis supervenes or the child may appear for a short time to completely recover from them, only to relapse when paresis occurs.

Their severity of course differs vastly, and while some children are extremely ill, others suffer from

little else than a slight malaise.

Unless an epidemic be in progress even expert medical observers may be entirely misled, as the febrile disturbances usually present may portend the onset of any of the acute infections of childhood. In some outbreaks the symptoms are mainly referred to the respiratory tract, in others to the gastro-intestinal.

The following are amongst the most common signs of an impending attack -

Fever is pretty constant and varies from a slight rise to a temperature of 103°F. or more. There is rarely any preliminary rigor. The temperature usually persists into the paralytic stage and subsides a few days later.

Sweating, often profuse in children is sometimes seen but is not by any means a constant accompaniment.

Drowsiness is usually noticeable, the child is listless and apathetic and does not wish to be disturbed If disturbed he is irritable and this irritability may be very marked.

Hyperaesthesia is an almost constant symptom and has been remarked by most observers, especially Müller in the Hesse-Nassau outbreak.

When moved the child cries loudly and appears to be very distinctly tender when touched.

Flexion of the neck and bending of the spine by

flexing the leg at the hip joint (Kernig's sign) causes acute agony.

There may be some slight rigidity of the neck muscles but it is rare to find a definite head retraction. The explanation of this pain on moving the spine is probably that in this stage there is a very acute inflammatory involvement of the meninges. It is difficult on account of the early age at which the disease occurs to ascertain whether or no the child suffers much spontaneous pain, but in those cases where the child is old enough to appreciate such things, this symptom is frequently complained of and appears to be usually situated in the neck, back or legs.

It has been suggested previously that such pain may be due to involvement of the posterior ganglia. It is not uncommon to find in this stage a preliminary weakness in one or other limb; the child may "go off his feet" or refuse to use one arm.

On examination no definite paresis is found and the tendon reflexes are neither constantly lost nor exaggerated. Perhaps the most frequent finding is that of an exaggeration in the early stages and a diminution or complete loss as paralysis supervenes.

Muscular twitchings and general convulsions are rare prodromata

Respiratory symptoms. These may if present vary

from a slight coryza to a marked broncho-pneumonia. In some a persistent nasal discharge has been noticed, whilst in others a reddening of the fauces and tonsils. A more common preliminary is a gastro-intestinal attack. There is often vomiting and loss of appetite and this may be accompanied by diarrhoea as noted especially by Krause[49a] in the Westphalian epidemic.

Other observers lay more stress upon a preliminary constipation.

On reviewing the above clinical picture of the prodromal stage, one is struck by the absence of definite characteristic signs, for there is no symptom of specific diagnostic value; perhaps the hyperaesthesia is the most constant and characteristic of the list.

Several other observers have hinted that the blood picture in the early stages may help one to come to a diagnosis, but their observations are extremely inconstant. Müller suggests that there is a leucopenia with relative lymphocytosis. La Fetra[50] on the other hand describes a leucocytosis, so when two such distinguished observers are at variance there is little to help us.

Examination of the spinal fluid in early cases is however, of the utmost importance. This will be dealt with at a later stage.

THE ACUTE STAGE.

This is an indefinite period but may be taken to start when the paralysis supervenes, and to terminate when tenderness disappears, and it is possible to flex the head without causing pain.

As may be well imagined from the diverse pathological findings the clinical picture is extremely varied.

In some children when paralysis comes on, the child is apparently partly stuporose. He lies on his back with his legs everted and thighs partially flexed in what has been aptly described as a frog-like attitude. He is easily roused and when an attempt is made to examine the affected limb, he tries to remove it from the examining hand by twisting about in bed and using the muscles that remain active. He is usually extremely fretful and cries on the slightest provocation.

Other cases (where meningeal involvement is more marked) shew a different attitude, namely, one in which there is a slight degree of head retraction, the child preferring to lie on one or the other side with knees drawn up (as in a tubercular meningitis). The child is extremely apprehensive of the approach of anyone to the bedside and will often cry long before he is touched.

Paralyses of the ocular muscles are not common, and the eyes react normally to light and accomodation.

The tongue is frequently furred and the throat and fauces slightly congested. Some observers have noted

an enlargement of the tonsils, Von Wiesner[51] quoted by Trethowan, believing that people with enlarged tonsils are very susceptible to the disease. A common accompaniment is that of enlargement of the glands in neck, axillae and groins.

An important symptom is that already mentioned, i.e. some rigidity of the posterior muscles of the neck; this is fairly constant but in slight cases may only manifest itself by a slight uptilting of the chin as the child lies in bed, and unless carefully looked for may be entirely missed.

In the vast majority of cases the chest and abdomen show no abnormality, but occasionally where there is paralysis of the abdominal lateral muscles or recti, a curious phenomena occurs, i.e. a somewhat alarming swelling is seen which enlarges considerably when the child cries; this may lead to confusion unless kept in mind.

The spleen and liver are usually normal in size.

Sometimes pain is complained of for some hours before the onset of paralysis but in many instances such is not the case, e.g. when a child may have been noticed to move a limb painlessly some hours previously it is now found to be quite paralysed.

It is a difficult task in the very young to map out definitely the muscle groups affected at an early stage, as the pain on moving is often so excessive that the

child will voluntarily prevent any motion of his limbs. These cases may be differentiated sometimes by the judicious use of a pin, for here the child will instinctively draw away the limb when he feels the prick.

Paralysis of the back muscles is a frequent occurrence but at first is difficult to demonstrate as the child will refuse to sit up from the pain caused by movement.

When the lower extremities are affected the knee jerk usually disappears in the injured limb, but not always so, and in some cases it is exaggerated. The loss of deep reflex is of not nearly so much importance as its reappearance when the paralysis is passing off: this return may be one of the first signs that power is coming back.

Profuse perspiration is not an uncommon symptom, but in many cases this hyperidrosis is slight or even absent. Cutaneous eruptions are not common. Retention of urine has been described but is rare; it usually only lasts for a few days.

The state of the bowels is important. Constipation is the rule.

Loss of sphincter control is not of common occurrence but does occur in some cases.

It may be definitely stated that pain in some form or another is constantly present.

It may be spontaneous, or only present when the limbs are moved or handled, and when pressure is exerted. The latter is much more common than the former, and seems to be most marked when the child's spine is flexed, e.g. by bending forward the head or by testing for Kernig's sign (i.e. flexing the thigh on the abdomen with the knee extended). These movements often produce intense agony to the small sufferer. Resistance to the performance of Kernig's movement is often largely voluntary on the patient's part, for thereby he prevents the vertebral bending. This symptom persists sometimes for many weeks and while present it is impossible to get the little one to sit up.

Spontaneous pain when present is probably in the form of a neuritis, and in older children and adults the pain may be so described that a nerve trunk inflammation is clearly indicated. This pain usually disappears in from 1-2 weeks.

Pain on pressure is not due to a hyperaesthesia of the skin but to muscle pain, for it is only elicited when the muscles are pinched.

Fever is usually present in the early stage, but is not often high and usually abates early. The fall is by lysis occupying from 12-24 hours.

It is important to note that in some cases the paralysis is apparently the first symptom of the disease

there being no prodromal period.

Another point to remember is that in some instances there is a definite remission of symptoms in the acute stage, the child appearing to get much better, only a few days later to suffer a relapse.

Cases in which deep stupor is present are not common but when they do occur are very easily confused with tubercular meningitis.

The patient lies in a semi-comatose state, but can usually be easily awakened, only to sink back again when the disturbance is removed. The stupor usually clears up rapidly.

The change from the acute atage to complete health is usually slow at first, but once started progress is fairly rapid, and later the child looks perfectly normal in everyway until the bedclothes are removed and the wasted, paralysed limbs are brought to light.

PARALYSIS.

The paralysis is not the one and only important symptom as was thought to be the case until recently, and to gain any control over the progress of the disease one must diagnose it early, i.e. before paralysis appears at all.

The paralysis ought to be confirmatory evidence of a previously suspected diagnosis. It is at present, however, the most prominent feature of the disease, but is by no means synonymous with it; viz. the abortive cases. It must be remembered that the clinical signs do not pepresent with any accuracy the pathological lesions.

The involvement of the cord is usually far more extensive than the muscular palsy would lead one to believe. This may be due to a very slight involvement or to the fact that most muscles derive nerve fibres from several sections of the cord. Disease in the white matter usually causes no characteristic physical signs, this no doubt being due to the fact that the grey matter is generally affected also: thus when there is lower neurone involvement the upper neurone disturbance is masked. The lower neurone picture in these cases predominates i.e. a flaccid paralysis is the most marked sign.

It is characteristic of poliomyelitis that the

paralyses are most diffuse and unsystematic in distribution, in one case an arm and leg are involved, in another there is a deltoid palsy and peroneal involvement, curious combinations with as far as one can tell at present no anatomical or physiological basis.

Of course, as is well known, certain sections of the cord are more often involved than others. The lumbar is the most frequent and the cervical next in order. The view most generally held is that this preferential site is due to the more abundant blood supply to these parts, for the first cellular infiltration takes place around the vessels, and consequently the extent of the inflammation will vary more or less with the number and size of the blood vessels.

The fact that the anterior horn is more frequently affected that the posterior grey matter or the white matter is probably also explained by the great vascularity of this area.

Paralyses are of two kinds - (1) Permanent and complete, (2) Transitory. In the former there is a greater destruction of nerve cells, probably extending over a large area, for as we have already noted most muscles are innervated from several nerve roots, and if only a smaller number of the cells giving origin to these axons are destroyed the probability is that the muscle will recover to some extent its power and usefulness.

In the second class, i.e. of transitory palsy the lesion may be one in which there is pressure exerted on the nerve cells by the oedema, exudate and haemorrhage, causing temporary loss of power, which, however, returns when this pressure is removed by the absorption of the exudate.

The onset of the paralysis is usually sudden and complete. A patient may fall asleep and wake up paralysed. There is seldom any great progression of the paralysis after the first stroke, but occasionally groups of muscles are rendered useless one after the other, and in one type of the disease (which simulates Landry's palsy) it spreads upwards and involves by degrees the whole cord, eventually causing death by destruction of the respiratory centre.

It has been noted that the legs and arms are most frequently affected, but it is interesting to take account of the relative frequency with which various muscle groups are involved.

The following table compiled by Lovett and Lucas[52] is of interest:-

1. Both legs.....130 cases.
2. Right leg.....216 "
3. Left leg.....239 "
4. Right arm..... 5 "
5. Left arm..... 5 "

6. Both arms alone.....0 cases

7. All four extremities..... 3 "

8. Arm and leg same side.....15 "

9. Arm and leg opposite side..... 7 "

10. One arm both legs..... 2 "

11. Abdomen with other paralyses..... 6 "

Let us now note the various paralyses more in detail, and differentiate between those affecting the cord only and those with also an implication of the bulbar region.

Spinal Paralyses.

Legs. As will be noted in the table reproduced above, the paralyses shew a decided preference for the lower limbs and in 50% of cases the legs are the only parts affected. This of course is due to an involvement of the lumbar enlargement of the cord, especially between the 1st lumbar and 2nd sacral segments.

An individual muscle group may be attacked or the whole limb involved. The former is the more usual and it is very frequently the Quadriceps Femoris that loses its function; this gives the characteristic negative knee jerk. In the lower leg the anterior group of muscles is more frequently injured than the posterior and it is usual to find a loss of power of the

anterior tibial muscle or peronei, or in the flexors of the foot and extensors of the toes.

It is an extraordinary but undoubted fact that it is rare to find a paralysed leg in which the toes cannot be flexed; or if at first affected they soon seem to regain their power. The injured limb is usually of much lower temperature than its neighbour and occasionally a purplish rash is present.

Paralysis of the sphincters is not a common occurrence: in many instances the children are so tiny that the passage of urine and faeces under them is a normal action, and it is difficult to estimate the amount played by sphincter flaccidity.

In a few instances there is a temporary retention of urine for a short time, but this speedily passes off.

Arms.

These are attacked next in frequency to the legs, the lesion being situated between the fifth cervical and first dorsal segment. The palsy is usually unilateral and in most cases there is also a simultaneous paralysis of one or both legs.

The tendency of the disease to pick out muscle groups holds also in the upper extremity: in the upper arm the deltoid and other shoulder muscles, the biceps and brachialis anticus. In the lower arm the elbow, hand and

finger muscles may be attacked. The flexors of the fingers (as was also seen in the case of the toes) often escape or when involved recover quickly.

For the whole upper limb to be permanently withered is rare. It has been noted that the proximal muscle groups in the limbs are more frequently attacked that the distal, and also that when recovery occurs it is earlier and more complete in character in the distal groups.

Diaphragm.

It is not frequent for the diaphragm to be attacked except in those cases which terminate fatally: in these latter death is almost always due to respiratory embarrassment and that means intercostal as well as diaphragmatic paralysis.

The reason that this large muscular structure so often escapes is that its innervation by the phrenic nerve takes origin from at least three cervical segments the third, fourth and fifth cervical: its numerous roots are probably its salvation, when respiratory involvement does occur the intercostal muscles are usually attacked first.

Cases have been noted where diaphragmatic paralysis has not resulted in death and where recovery has taken place.

The typical picture of diaphragmatic palsy is as

follows:- Respiration is wholly thoracic and at times the accessory muscles of respiration are called into play. The abdominal wall moves, but on inspiration there is retraction instead of protrusion.

This was noted in the fatal case recorded at the end of this paper.

Intercostal.

Invasion of the dorsal section of the cord causes paralysis of the intercostal muscles. This is extremely serious ~~later~~ but is not necessarily fatal though usually so.

In early cases of this type one notes that respiration is entirely abdominal, the chest moving very slightly if at all. Sometimes there is sucking in of the intercostal spaces, later if the child recovers marked atrophy is noted of the muscles. The chest appears narrow and the abdomen abnormally prominent. Pulmonary oedema is frequent and broncho pneumonia not uncommon.

The abdominal muscles may be paralysed in toto or locally. In the latter case we see the hernia like bulgings which we have already noted.

The muscles of the neck and back are often affected early but tend to recover quickly. The extent of the lesion is difficult to estimate especially in acute

cases when pain is such a prominent symptom.

Where the neck participates, the child is unable to hold up the head and it rolls helplessly about when any attempt is made to do so. The child cannot sit up when the back muscles are affected.

Bulbospinal Paralysis.

We have already seen that there is much to lead one to believe that in poliomyelitis the pathological process is often much greater than is suspected by merely studying clinical symptoms. There may be exudate and haemorrhage present in the medulla and pons but these will only give rise to symptoms if involving the cranial nerve nuclei. When involvement of these nerves takes place there is usually a simultaneous process in the cord, but not always, and in some instances evidence of a purely bulbar palsy is all that can be found.

The affections of these cranial nerves are mostly unilateral.

It is quite impossible to draw any definite line of demarcation between the spinal and the bulbar as they so frequently merge the one into the other.

These cranial nerve palsies are often transient and slight in character and may easily be missed. It has been stated that when associated with cord lesions the outlook is not nearly so grave as when they occur alone.

Of all the cranial nerves affected the facial is the most frequent: it may be injured in toto or only one branch be touched. The ocular nerve is also paralysed comparatively frequently, and these ocular palsies vary from the affection of one muscle to a complete ophthalmoplegia.

The optic nerve is rarely if ever involved.

Speech, phonation and deglutition may all be interfered with from implication of their respective nerve centres.

The prognosis is usually good and recovery frequently occurs.

In these lesions of the upper neurone when the child begins to get about again, an ataxia is frequently noted with increased knee jerk and spastic gait. Its cause is by no means easy of explanation.

DIAGNOSIS.

As we have already seen it is extremely difficult if not well nigh impossible to diagnose the sporadic form of poliomyelitis before the paralysis sets in. The premonitory symptoms are so confusing and are common to so many diseases of infancy that the problem is only solved after careful examination and by adopting a process of exclusion.

When, however, the palsy has supervened the riddle is usually clear, but there are several diseases not unlike poliomyelitis that may still lead to an error of diagnosis.

These must be kept in mind, especially the following:- The pseudo-paralysis of rickets, scurvy, infective epiphysitis, acute rheumatism and tubercular disease of the knee and hip. Purves Stewart in his book on the "Diagnosis of Nervous Disease" mentions that form of muscular atrophy known from its distribution as Tooth's peroneal type or as the progressive neurotic atrophy of Charcot and Marie.

This disease comes on in childhood and attacks the peripheral parts of the limb, producing weakness and contraction. It is hereditary and runs in families. It usually comes on slowly. The most characteristic deformities caused are those of claw-foot and claw-hand,

these in young children the writer states, to be almost pathognomic of the disease. Deep reflexes are lost in the atrophied muscles. The pathology seems to be an atrophy of anterior cornual cells without degeneration of anterior nerve roots; the peripheral nerves supplying the affected muscles are however diseased.

The same author notes another nervous disease which may be confused with poliomyelitis. This is Interstitial Hypertrophic Neuritis. It occurs in childhood and adolescence and gives rise to a flaccid muscular atrophy with sensory changes, these later, take the form of shooting pains, anaesthesias and analgesia. There is ataxia, loss of knee jerk. The Argyll-Robertson phenomenon is present and there is frequently kypho-scoliosis.

It is unlikely that mistakes will be made in diagnosing these from poliomyelitis but it is just as well not to forget their occurrence.

Cases which prove fatal.

The Rapidly Progressive Cases are most distressing. In this type the disease does not suddenly put one group of muscles out of action and then discontinue its attack, as is usually the case in the more ordinary type, but here the paralysis is progressive and slowly creeping over the body, finally involves the muscles of respiration and the patient dies of suffocation. Death in acute anterior poliomyelitis is due in the vast numbers of cases to this progressive type or to a complication, e.g. broncho pneumonia, which attacking a devitalised system proves extremely virulent.

It is usually necessary for both the intercostal muscles and the diaphragm to be paralysed for death to occur, for if only one of these be affected patients can survive.

The patient is often fully conscious and his senses are keen and alert till the end, and this rather disproves the assertion that it is a toxaemia that overcomes him.

Taking a rough average of the various epidemics, the mortality appears to vary from 10% to 20%.

Landry's paralysis is of this group and is an ascending paralysis involving first the legs, then the arms, intercostals, neck and diaphragm.

The fatal cases usually die in a few days and after

the acute onset there may be a pause before the progress continues. The first evidence may be difficulty in deglutition or rapid respiration, this gradually gets worse and the patient has to fight for breath, using his alae nasi and accessory respiratory muscles. The condition of the lungs is marked, there are coarse moist rales heard, shewing that oedema is present.

There is frequently no change in the heart, its beat is strong and regular till it stops.

In these fatal cases there is often more marked fever than in those in which recovery occurs.

Sweating is profuse, pallor is the rule with perhaps a slight bluish tinging of the lips.

Cerebral Types.

It is a fact that the virus of acute anterior poliomyelitis may cause symptoms of cerebral rather than of spinal origin, but it is still a debatable point as to whether a true polioencephalitis exists. It was in 1885 that Strümpell(53) noted an analogy between certain forms of cerebral paralysis in children and poliomyelitis. It was

It was he who suggested the name Polioencephalitis he quoted 24 cases. There was an acute initial stage with convulsions, fever and vomiting, then after a short period a hemiplegia was noted. The limbs were more affected than the face. There was no atrophy or

or reaction of degeneration. Reflexes were ---. Exaggerated. To quote his own words "in both diseases the chief seat of the lesion is in the gray matter, in one case in the gray matter of the anterior horns and in the other in the corresponding portion of the cerebrum".

The first full account of the epidemiology of this disease was published by Medin(54) in 1898, and he included reports on three cases shewing cerebral symptoms, spastic hemiplegia and exaggerated reflexes. He was convinced that these cases were caused by the same virus as that known to cause acute anterior poliomyelitis.

Confusion has arisen with regard to this type of disease, as many writers refer to cases as cerebral, etc. when certain symptoms, e.g. stupor, delirium, etc. have occurred during the attack.

There is no definite hard and fast line between the cerebral and the spinal cases; the bulbar affections form a connecting link between the two. The two types merge clinically and pathologically. The best way seems to be to take an anatomical basis, and it would be advisable to judge by the most prominent symptom, e.g. a case with spastic hemiplegia would undoubtedly be cerebral, and again one with flaccid face and arm paralysis - bulbospinal.

To prove definitely that the virus be the same in cerebral and spinal cases experimental evidence is necessary, and so far this has not been convincing;- as yet

the virus has not been shewn to exist in typical polioencephalitis by transference of the disease to monkeys, and it is noteworthy that although monkeys are injected intracerebrally, the resulting paralysis is always spinal and never cerebral.

Anderson and Frost(55) note a case where the blood serum of a patient who had a spastic paraplegia of the legs, neutralised active virus.

The chief evidence in favour of the two diseases having a common origin is the fact that (1) these cerebral spastic cases are found in true epidemics of poliomyelitis, (2) that flaccid and spastic paralyses may occur in the same family or even in the same patient during an outbreak of this disease. The latter fact has been recorded by two observers, Möbius(56) and Hoffmann(57).

But against this it must be remembered that in spite of the more general recognition of poliomyelitis in late years, there has been no corresponding increase in the number of reported cerebral cases.

It is a significant fact that Wickman who probably has had more experience of the disease than any other living observer, states that in the great Swedish epidemic of 1905, there were no true cerebral cases.
If we take the anatomical basis for our classification we see that true polioencephalitis is extremely rare, if it occurs at all.

Abortive Cases.

A few words must be said about the important group of abortive cases, i.e. those in which infection occurs but paralysis does not develope. They are very contagious and as before stated in this discourse, very easily missed. An important discovery was made by Netter and Levaditi[58] when they demonstrated that the serum of abortive cases can neutralise the active virus in vitro.

This test is of course confirmatory. Other points which are also of importance in the diagnosis are - history of exposure, clinical picture, blood and cerebro spinal fluid examinations.

The clinical picture is indefinite; there is frequently drowsiness and irritability: the reflexes may be exaggerated or absent: pain is common either in the neck or head. The disease is likely to be confounded with gastric trouble of influenza.

There is little help to be got from an examination of the blood, as different observers give such conflicting reports. One side contend that in poliomyelitis there is a leucopenia; the others say a leucocytosis, and this latter class quarrel as to whether the increase is ~~of~~ lymphocytic or polymorphonuclear.

Cerebro-Spinal Fluid.

We now come to an extremely important aspect of the study of this disease, namely the careful examination of the cerebro-spinal fluid. First, it may help to clear up a doubtful diagnosis in early cases, and secondly it will shew how the disease is progressing under treatment.

Laborious experiments have been made by Wollstein[59] and Römer and Joseph[60] to demonstrate the presence of a specific antibody in the spinal fluid, but in vain.

The naked eye appearance is generally as follows - The fluid is clear and colourless, in some cases on standing a cobweb-like reticulum is found.

Pressure is usually excessive. Chloride estimation proves that these inorganic substances are normal in quantity. Reduction of Fehling's solution takes place as in normal cerebro-spinal fluid. (This is important as in many cases of meningitis the reducing power is lost).

Mononuclear cells predominate in the fluid, except in the very early stage when they are outnumbered by the polymorphs.

Flexner and Lewis [61] studied the cerebro-spinal fluid of an inoculated monkey at intervals, and the result was as follows:-

"Twenty four hours after inoculation a considerable

number of small cells were seen hardly exceeding a lymphocyte in size but with a polyform nucleus, a few lymphocytes and some red blood corpuscles. At the expiration of the forty eight hours, the white cells have increased in number, but the cells with polyform nuclei still predominate. At the expiration of seventy two hours, a large number of mononuclear cells have appeared and the fluid presents a striking opalescent appearance. On the day of paralysis the fluid tends to be only slightly cloudy and contains a mixture of large and small (lymphoid) ~~neurone~~ mononuclear cells and a few cells with polymorphous nuclei".

If a cell count is made immediately after the fluid is obtained, one will find in a normal fluid from 3-12 cells per cubic millimetre. In poliomyelitis we find a vast increase on this figure, in general the highest cell counts are found in the early days of the disease and there is a progressive falling off as time advances: the count may shew many hundred cells per cubic millimetre.

In contrast to the cell count the globulin content is usually low in the first part of the acute stage. It rises during the second and third weeks and then gradually falls, though frequently globulin persists long after acute symptoms have passed off.

The globulin content is best tested by the butyric acid method of Noguchi. To 0.2 c.c. of spinal fluid is added 0.5 c.c. of butyric acid solution (10% butyric acid in 0.85% solution of sodium chloride). The mixture is boiled, 0.1 c.c. of normal sodium hydrate solution added and then boiled again.

The fluid is allowed to stand for 5-10 minutes, when if positive a precipitate forms. When faint this indicates a slight reaction, when heavy and flocculent - a marked one.

Perhaps taking a series of cases the commonest type of fluid seen is one with a slightly raised cell count and a well marked globulin reaction.

There are, however, two other distinct types seen. One is a fluid with a high cell count and a normal or very slight globulin reaction, and the other is a fluid with a normal or low cell count and a very marked globulin reaction.

Each type of fluid is however, quite definitely characteristic of a stage of the disease. The cellular exudate is almost always associated with the earliest days of the acute stage; the albuminous exudate with the latter part of the acute stage.

As regards early diagnosis the cases that can be examined before the onset of the paralysis are of the greatest interest.

Where this has been done the fluid generally shows -

1. A great excess of cells per cubic millimetre, reaching maybe to 700-800.

2. A predominance of the polymorphonuclear cells - 80-90%.

3. A slight globulin reaction.

Fissell[62] described a case where early lumbar puncture suggested poliomyelitis and his result was similar to the above but without the polymorphonuclear excess.

The diagnosis of poliomyelitis cannot be definitely made from an examination of the spinal fluid, for the organism, if such there be, is ultra-microscopic, and exhaustive biological research has failed to detect the presence of antibodies in the fluid. On the other hand, an examination of the cerebro-spinal fluid in cerebro-spinal tubercular or pneumococcic meningitis is usually diagnostic, for the causal organism can be found.

The failure to find organisms in a fluid with a polymorphonuclear excess would at any rate be suggestive of poliomyelitis.

Although the lumbar puncture and examination of the cerebro-spinal fluid is not of specific diagnostic value it is of extreme importance, for it helps by ruling out possibilities of other diseases, which may be confused clinically in the pre-paralytic stages with poliomyelitis

Lumbar puncture is an entirely safe proceeding, and should be used more widely than it is at present, for the value of any future method of treatment of poliomyelitis must depend on the possibility of early diagnosis, for where the nerve cells have been destroyed the results from any therapeutic invasion will be comparatively small.

Let us sum up the points which are of value and help in making an early diagnosis from examination of cerebro-spinal fluid.

1. For several weeks after the onset the fluid usually deviates from normal.
2. Early cases usually shew an increased cell count with normal or low globulin content. Here also polymorphs are usually in excess, but often large mononuclei and lymphocytes preponderate.
3. After the first two weeks , cell count drops to normal and the globulin content is increased.
4. Fluids reduce Fehling.

It must be remembered that abortive cases shew analogous changes.

Prognosis.

This must invariably be guarded: there is no disease where errors in prognosis are more common; for the problem is complicated by the paralyses.

The mortality rate is excessively high and has been variously estimated from 10% to 22.5%, the latter figure being given by Lindner and Mally(63). An average percentage would be about 15%. Batten(64) gives 12% but this does not help us much in individual cases. Age seems an important factor, the outlook is brighter in young children than in older ones or in adults.

The question to be answered if the case is seen in the pre-paralytic stage is "will paralysis occur or not"? We have nothing pathognomonic to guide us to an answer. The cerebro spinal fluid may help us to a definite diagnosis of poliomyelitis, but it will be unable to prognosticate a palsy.

The presence of pain in a limb will not guide one much, for pain in this stage is of such constant occurrence, also the presence or absence of knee jerk is of very little value.

With regard to the residual paralysis, here again there is little to guide us, and time alone will shew the amount of power that will return to a limb. The most surprising results do occur and it is extraordinary the way in which these cases repay patient systematic treatment.

Again, granted that paralysis be present, say on the second day, we are unable to say with any assurance whether it will progress or not, unless the case seems definitely to be assuming the characteristics of a Landry's type. Unfortunately we have no way of telling where the lesion will occur or if an existing lesion w will advance.

It may be stated that in most cases the initial lesion is the final one.

Death seems to be of more common occurrence when it is the cervical region that is affected; but it must be remembered that cells which lie just near the zone involved in the ~~path~~ pathological process may continue to functionate, and the phrenic and intercostal centres may escape entirely even when the lesion appears to be quite close to them.

Fatalities generally occur on the fifth to eighth day if uncomplicated. As before stated a patient can recover if only one part of the respiratory mechanism be involved, i.e. the diaphragm or thoracic muscles alone, but if both be affected death is certain. Even when one part is paralysed the outlook is extremely grave for pneumonic conditions are more likely to occur in these cases.

TREATMENT.

The problem in the treatment of poliomyelitis consists in preventing the spread of the disease to others; in applying general symptomatical treatment, and in trying to restore to the wounded musculature as much power as is possible with the least deformity.

We have as yet no specific form of therapy, nothing to prevent the occurrence or control the spread of the paralysis, and no means of helping resolution of the inflammatory process. The disease has undoubtedly been proved to be of an infectious nature and therefore isolation is essential, although it is difficult to convince people of this fact.

Notification and disinfection are required in Sweden and Germany, and recently the Local Government Board in England have in many places made poliomyelitis a notifiable disease.

The length of quarantine is not definitely fixed yet for the virus has been shewn to be present many months after an acute attack, but four to eight weeks is usually sufficient for ordinary safety. In British Hospitals the sporadic cases which are seen are usually treated in the general ward, and with little or no spread, but where epidemics occur the strictest precautions should be taken, and the patient treated in exactly the same way as any case of scarlet fever or diphtheria -

disinfection of sheets, bed-pans, fomites, etc.

Acute Stage.

The child must be kept in bed, in a well aired, quiet room and given a light but nourishing diet.

The bowels must be kept regular with some purgative of which perhaps the best here is calomel (gr.i-ii). The skin ought to act freely, this being brought about by a hot pack and a mild diuretic given (Pot.Citrate grains V. T.D.'S.

The most pressing symptom requiring treatment is the pain. This may be spontaneous but is usually not marked unless movement of the limbs is brought about. Great care must be exercised in the handling of these little ones as their agony may be intense, and movements calculated to increase their discomfort, e.g. flexure of head or neck, flexing hips, bending spine should be avoided as much as possible. The weight of the bedclothes is sometimes sufficient to produce pain and in these cases a cradle may help matters greatly. Light padded splints give comfort and support to the injured limb.

The affected members are cold and clammy, and then vitality should be sustained by wrapping them warmly in wadding.

Where these measures are not sufficient analgesic drugs must be resorted to, of these many have been tried.

Salicylate iii of Sodium (gr.ii) every 2 hours, sometimes gives relief. Bromide, aspirin and occasionally small doses of Tr.Opii (m i every 3 hours) are all of service. Where cerebral symptoms are prominent an ice-cap is useful and a lumbar puncture may be tried to relieve pressure.

Where intense poliomyelitic pain is present a belladonna and morphia suppository often acts like a charm. The composition of such a suppository would be -

Pulv.Opii.....gr.1/2

Ext.Bellad.....gr.1/8

Sod.Sal.....gr.V.

Oil.Theobron.q.s.....qs. Ft.Suppos.

One every 3 hrs. till pain relieved.

Urotropin (hexamethylenetetramin) is given credit for having an antiseptic action on the virus in the spinal canal. Flexner(65) strongly believes in its efficacy. He quotes Cushing who has proved that this drug is in part eliminated into the subdural space of the canal and consequently exerts some antiseptic action on the fluid.

Flexner supported Cushing's observations by showing that Urotropin can be demonstrated in the cerebrospinal fluid of monkeys some little time after being taken by the mouth.

He experimented with the substance and in a few

cases managed to get satisfactory results. Some monkeys having previously been treated with urotropin withstood an injection of anterior poliomyelitic virus and in others the onset of paralysis was prolonged for twenty four hours.

The dose is gr.iii-v. every 4-6 hours.

Ergot, has been recommended with the object of contracting the vessels of the cord and so diminishing congestion.

Potassium Iodide is given in the later stage with a view to removing the products of inflammation.

Experiments have been made by Clark(66) at the request of Dr. Meltzer, in order to test the action of subdural injections of epinephrin in cases of experimental poliomyelitis.

The view taken was, that if the lesions - especially ascending ones - be due to progressive vascular involvement with transudation and oedema, then contraction of the vessels by this substance would tend to limit the spread of the disease, for this response of the vessels should bring about a cessation of the exudation, and so prevent injury by the inflammatory oedema upon adjacent nerve cells.

This amelioration will of course only occur if the ascending paralysis be due to a consecutive vascular

involvement; if it should be caused by a continuous upward involvement of nerve cells then epinephrin will be of no use whatever. In his experiment Flexner injected the epinephrin into the subdural space of monkeys almost moribund with the disease, the amount used being from 1-1½c.c. of a 1 to 1000 solution. His results were not very convincing, but the general effect was an improvement of muscular tonus in the paralysed muscles and in the respiratory movements. In some cases life was distinctly prolonged for some hours.

Its efficacy is still subjudice, but it is quite possible that it might in human cases cause a limitation in the spread of the disease by its action upon the blood vessels and its consequent control of exudation.

Further work is necessary before any definite statement can be made.

Counter irritation of the spine is of doubtful value. Some clinicians continue to use it, but Sachs(67) thinks it has little or no effect, favours bedsores and increases the patient's pain.

As soon as the pain passes off and the part can be safely handled, treatment must be directed towards the restoration of muscular function and the prevention of deformities.. Contractions develop early in poliomyelitis and must be guarded against from the onset. Foot drop is a good example of this, the pressure of

the bedclothes increasing the deformity. Talipes shoes sandbags, and cradles are all useful in the prevention of this overaction of the sound muscles.

Any apparatus must of course only be applied in the intervals between active and passive movements.

Massage.

This is of great use and must be commenced as soon as the pain subsides. It must be given daily and continued for many months. It assists the circulation and replaces the lost muscular activity.

With massage passive motion is commenced, i.e. the rhythmic performance of certain movements and stimulating the patient to try and attempt them himself.

The active method is a great education for the muscle and is perhaps the most useful of all methods of treatment. The patient must be persuaded to persist in making attempts at movement, often a difficult task especially in the very young, and much ingenuity must be exercised before it is accomplished.

The movements may be assisted by their being carried out in a warm bath. The patient must be recommended if the arm be involved, to move his fingers and touch objects, move his wrist, elbow and shoulders.

Similar exercises must be devised for the lower limb, and he must try to sit up from the lying position.

When he can walk a little he is helped greatly at first by some slight support, e.g. a go-cart or wheeled chair to push along the floor.

These exercises and movements must be persevered with for quite 18 months to 2 years, for often improvement continues all the time.

Electricity helps, the galvanic current being used

Splints, braces, etc. all play their part in the prevention of deformities, but must be discontinued as soon as possible, for if mechanical supports be continued too long, the patient will come to rely too much on these and cease developing his muscles to their utmost.

When contraction and deformities have occurred the surgeon is requisitioned and to go into the technicalities and details of nerve anastomosis, muscle transplantation, tenotomies, ankylosis, wrenching, forcing, plaster of Paris ~~casts~~ of cases etc. is outside the scope of this work.

For Prophylaxis contacts must be isolated, given small doses of urotropin and a H_2O_2 nose wash and gargle. Streets watered and flies exterminated.

In conclusion continue and persevere with treatment, for although it is slow and laborious work, the result is often many times more satisfactory than one would ever have dreamed.

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NOTES ON CASES.

CASE I.

E.Y. Male. Aged 4 yrs.

Admitted to the Childrens' Infirmary, Liverpool, July 11, 1912. Discharged Dec.31, 1912. Result - Improvement.

Family History.

Parents and 4 other children strong and healthy. One child suffers from rheumatism. Phthisis and heart disease in mother's family. She had one miscarriage.

Previous Illness.

Chickenpox and whooping cough when a baby. Pneumonia at 1 year. Rickets 1½ year.

Present Illness.

On July 1st (ten days previous to admission) child seemed languid and feverish: his throat was slightly sore and there was some abdominal pain and pain in the back of the neck.

Four days later (July 5th) it was noticed that he was quite helpless and could not move either arms or legs. He was also absolutely unable to sit up in bed.

His eyes rolled and his mouth showed a tendency to twitch. He was extremely restless and talkative in his sleep. Appetite poor. Bowels very constipated.

On Admission.

General Inspection, etc. His general appearance is that of a well nourished child, but there is some facial pallor. The mucous membranes are well coloured. Teeth good. Tongue foul and coated. Eyes - pupils are slightly unequal, the left one being the larger. The child is not irritable but is extremely tender to pressure.

The skin is excessively moist. He lies quietly in bed with the lower limbs in a frog-like attitude.

There are no enlarged cervical glands but in the groin and axilla a few glands can be felt. There is no evidence of syphilis, rickets or tuberculosis.

Temp. 98.8. Resp. 28. Pulse 108.

Nervous System.

The head is well shaped. There are no spinal curvatures and no head retraction is noted.

Mental Condition.

Child is quite conscious and intelligent, answers questions.clearly.

Motor Functions.

The musculature is generally good. There is no obvious wasting of any group as yet.

The upper and lower limbs appear to be almost paralysed and cannot be moved at all voluntary with the exception of the toes, fingers and ankles which seem to have escaped.

There are no involuntary movements, spasms or convulsions. The child is quite unable to sit up, all the back muscles being paralysed, but the head can be moved from side to side.

Reflexes.

The knee jerks and tendo achilles are entirely absent and the biceps and extensor jerk in the arm cannot be elicited.

Kernig -. Babinski -.

Sensory Functions

These appear unaffected. The child is however, extremely tender to touch, and even when approached ~~the~~ bed, a look of nervous apprehension appears, lest one be going to touch his tender limbs.

Lumbar Puncture.

Clear fluid - 20 oz. No coagulation. Albumen +. Fehling reduced. Lymphocytes present +- +.

Eyes.

The pupils are slightly unequal, the left being the larger of the two. They react freely to light and accomodation. No nystagmus. Fundi are normal.

Ears.

Normal. No discharge.

Digestive System.

Tongue foul. Teeth good. Child takes his food well. There is no vomiting. The bowels tend to be constipated. Fauces clear.

Abdomen.

Nil. No enlargement of organs, no lumps or fluid.

Respiratory System.

No alteration. Percussion note#.* Breath sounds normal, puerile in type, no accompaniments.

Heart.
No murmur or enlargement.

Genito-urinary System.
Urinates in bed apparently unconsciously. Urine contains no abnormal constituents.

16.7.12. Still absolutely helpless. Excessive thirst. Pespiration is very profuse. Temperature and pulse normal.

22.7.12. Slight improvement. Tenderness less. Can move wrists and fingers slightly.

26.7.12. Fingers easily moved. Still profuse pespiration. Some tenderness.

31.7.12. Can flex wrists. General condition improving. Incontinence of urine continues. Temperature and pulse normal.

6.8.12. On having right arm flexed he can keep it in that place for a short time. Left arm - no flexion possible. Legs can be drawn up with extreme difficulty

18.8.12. Can almost touch nose voluntary with right hand, flexing it at elbow. Massage commenced. Temperature and pulse normal.

20.9.12 Massage and galvanism. Development of papular rash on back and buttocks.

25.9.12. Back healing nicely. Left leg has some power: can just raise from bed.

21.10.12. Tendency to talipes of right foot. Talipes shoe applied. Right arm quite useful, slight flexion at wrist. Left arm powerless except for flexion and extension of wrist. Child cries with pain if back be extended. Knee jerks still entirely absent.

20.11.12. Improvement continues slowly.

30.12.12. To be discharged. Condition unsatisfactory as regards paralysis but general condition good. Pespiration disappeared.
Arms. Right arm - no movement possible at shoulder. Deltoid paralysed, considerable atrophy of right deltoid muscle. Biceps has recovered also triceps. Extensors and flexors of wrist now working: fingers also can be freely moved.

Left arm - considerable atrophy. All muscle groups paralysed from shoulder down except extensor of wrist and flexors and extensors of fingers.

Legs. Right - only toes of right leg can be moved. Considerable atrophy.
Left - can be lifted from bed and held suspended. Movement good at knee and ankle.

Back muscles. Can pull himself up by side of bed and can sit erect for a short time.

Treatment in brief consisted of large doses of urotropin. Later tonics, strychnine, cod-liver oil and sanatogen. Massage, passive movement and electricity.

CASE II.

C. Mc.B. Female. Aged 1½ years.

Admitted Oct.4th, 1912. Discharged Jan.3rd, 1913.
Result - Relieved.

Family History.
Parents and 8 other children strong and healthy. Phthisis on father's side. No miscarriages. Home surroundings good. Breast fed for 12 months.

Previous Illnesses.
Nil. Always healthy.

Present illness.
On Sept.29th (5 days ago) the child seemed languid and drowsy. The mother noticed some twitching of the face. She gave an aperient and the child became rather better.
On Sept.30th the child was very drowsy and took no interest in its surroundings.
On Oct.1st she vomited and was very feverish and restless all day. The face was puffy especially on the left side the lips were swollen and mouth and tongue very dry.
On Oct.3rd she became gradually unconscious and lost the use of arms and legs.
Bowels very constipated.

On admission.

General inspection etc. Weight 1st. 2lb. 3oz. The nutrition is good, the colour fair, skin moist. Child lies quietly in bed in a semi-conscious state but can be roused sufficiently to take her meals, drinking milk from a feeding cup. Is perfectly placid and does not even move her head from side to side.
Head well shaped. Facial irritability not increased. No enlargement of cervical glands. No evidence of rickets, syphilis or tuberculosis.
Temperature - 98°. Pulse 144. Respiration - 36.

Nervous System.

Head well shaped. Fontanelle slightly depressed. No head retraction. Some pain on extending neck. No spinal disease or curvature.

Mental Condition.

Child is extremely drowsy and seems semi-conscious. Can be roused with some difficulty to be fed. There is no excessive irritability.

Motor Functions.

There is a complete flaccid paralysis of both arms and legs, back and neck also appear to be affected. There is no wasting as yet. The legs lie widely spread out in the frog-like attitude.

There are no involuntary movemente noted, no tremors spasms or convulsions.

Reflexes - Deep. Absolutely abolished in both upper and lower limbs.

Superficial. Abdominal reflex is present but sluggish.

Kernig's sign is negative.

Brudsynski's sign is also negative.

Sensation.

Difficult to estimate but appears to be unimpaired.

Eyes.

There is marked ptosis of left eyelid, pupils are equal and react to light, but the left pupil reacts more sluggishly than its fellow.

Ears.

No discharge from either.

Lumbar Puncture.

Fluid is clear (30c.c.) Reduction of Fehling

normal. No coagulation after standing for 24 hours. Film. A few polymorphs present. Great excess of lymphocytes noted.

Further Notes.

12.10.12. Ptosis is rapidly disappearing. Child less drowsy, takes a little notice. Can move head from side to side.

14.10.12. Moves fingers of both hands.

18.10.12. Slight attack of ileocolitis, blood and mucus in stools, frequent motions. To have barley water. Rectal wash outs.

20.10.12. Stools less frequent, very little mucus.

27.10.12. Stools normal. The paralysis is making very little progress.

2.11.12. This morning the face is rather puffy and the limbs slightly swollen, some oedema. No albumen in urine but urine is acid.
On microscopical examination motile bacilli are found.
Cultures - pure cultures of Bacillus Coli found.

9.11.12. Has been on large doses of Citrate of Potash for a week and urine now alkaline. General improvement.

9.12.12. Limbs improving. Can use right arm a little, lifting it to her mouth. Can flex left forearm but cannot move shoulder.
Legs absolutely no sign of voluntary movement. Knee-jerks absent in both limbs. At times they still seem tender to touch. Face shows no paresis now.
Urine kept alkaline.

27.12.12. Can just draw up legs a little. Weight - 1 st. 4lbs. 4oz.
Urinary condition almost cured. Pathological reports no B.Coli. Urine alkaline, contains few staphylococcus albus. Child is to go home and come up to Out Patients' Department for massage and electricity.

N.B.
Temperature never rose above 99° all the time she was in hospital. She gained weight steadily from admission to discharge.

Treatment.
Urotropin Vgr. T.D.S. and later for coli cystitis

Pot.Cit. gr.XV every 4 hrs, gradually reduced to V grs.
T.D's.
Tonics - Codliver oil, etc.
Massage, active and passive movements and galvanic current.

CASE III.

J.S. Female. Aged 1year,3 mos.

Admitted Dec.3rd, 1912. Died - Dec. 21,1912.

Diagnosis - Polioencephalitis.

Family History.

Both father and mother specific. One other child died when 3 weeks old.

Previous History.

Full time, healthy baby. When 3 weeks old developed rash, first on buttocks then spread over body ? syphilis. Taken to doctor and attended for 3 months. Child then well until July 12th 1912 when she was suddenly taken ill with convulsions lasting for several hours: feverish and vomiting.

The mother noticed that she could not use the right arm and could not sit up, though she had previously done so quite well.

Seen in Out Patient Department next day but refused to leave the child in hospital.

Not seen again until Dec.3rd, then said that on Nov.29th child had again been feverish and vomited: sent up by doctor and admitted.

Breast fed. Always very constipated.

Out Patient Department Notes. July 16th, 1912.

Mother says child had fit 10 days ago and was feverish and vomited. She has lost use of right arm and is unable to sit up. She is also extremely tender to touch.

There is considerable weakening of right arm, especially of the extensors of the wrist - wrist drop. Face and legs unaffected. Arm flexed and adducted. Thumb across palm unable to grasp any object. Considerable tenderness. Knee jerks + +.

Told to return in 1 week. Not seen again until Dec.3, 1912, when condition was as follows:-

Dec.4th, 1912. On admission.

General condition. Development good; fair colouring, no anaemia or jaundice. Skin moist but no excessive pespiration. No eruption or oedema. Lies comfortably in any position and cries lustily when disturbed. Temperature 98.4°. No definite evidence of rickets, syphilis, etc.

Nervous System.

Head circumference 18", slightly square in shape, fontanelle open; no bulging.

Spine normal, no curvature or tenderness.

Mental Condition.

Irritability. Sleeps well but not abnormally drowsy.

Motor Functions.

Marked paralysis of left face involving whole side. Eyelid cannot be closed properly and there is excessive lachrymation.

Also paresis of right arm especially the lower arm. Cannot grasp objects but can flex forearm and raise shoulder. Limb colder than left one. The paralysis is spastic in type. The right leg can be moved slightly. No involvement of abdomen or respiratory muscles.

No involuntary movements (no spasm tremor or convulsions).

Voluntary Movements.

Child cannot sit up or hold up its head.

Reflexes. Greatly exaggerated especially knee jerk.

Eyes.

Paralysis of left orbicularis. Pupils unequal. Left pupil larger than right and reaction to light is sluggish.

Digestive System.

Tongue dry and foul. Teeth 6 in number. Fauces clear. Appetite poor. Vomits feeds. Bowels constipated. Abdomen - soft, no lumps, no enlargement of organs, no free fluid.

Respiratory System.

Shape of chest normal. Bronchial sounds vesicular no accompaniments. No alteration in

Circulatory System.
Heart - no enlargement or endocardial bruit.

Haemopaietic System.
Spleen not enlarged. Lymph glands in neck. Axilla and groin not palpable.

Genito-urinary System.
No difficulty in micturition. Urine - nil.

Further notes.
9.12.12 Condition much the same. Takes well. Bowels normal. Temperature normal.

14.12.12 Temperature 99.4°.

17.12.12 Examination of fundi. Discs normal. Temp.97°.

18.12.12. Child extremely irritable. Conjunctivae injected. Skin elasticity less. Colour earthy. Rapid pulse. No Kernig. Losing weight. Pupils still dilated with atropine. Temp. 97°.

19.12.12. Unconscious. Pupils dilated (? atropine), do not react. No Kernig. No Brudsymski. Doughy abdomen. Spasticity increased. Knee jerks +. Temp.96.4°. Unable to swallow. Nasal fed.

20.12.12 Coma deepened. Died 12 p.m. Unfortunately no permission for P.M.

CASE IV.

A.T. Male. Aged 3½ years.

Admitted to the Children's Infirmary, Liverpool, March 10th, 1913.

Complaint - paralyses of legs and one arm.

Family History.
Parents are both in good health. Three other children alive and well. Mother has had no stillborn children and no miscarriages. Birth absolutely normal, no instruments used.

Hygienic surroundings - Satisfactory.

Previous Health.

Child has had no previous sickness, with the exception of an attack of bronchitis some 6 months ago.

Present Illness.

Eleven days before admission (Feb.28th,1912), patient was perfectly well. On the morning of March 1st he awoke in a rather feverish state and complained of severe abdominal pain. He vomited once after his early meal. He was also noticed to be rather fretful. No cause could be assigned for this malaise.

Later in the morning he was sitting on the floor and when told to rise, it was found that he could not do so; the parents naturally became alarmed and tried to make him walk but all to no purpose.

He was very fretful and when handled screamed with pain. He was put into bed and the doctor sent for. He was given some opening medicine and appeared a little better later in the day.

The next day (March 2nd) the condition remained much the same and although the legs appeared to be quite helpless he could use both arms. He would not however, sit up or hold up his head.

March 3rd found him as before, but now his parents noticed that he could not use his left hand and the arm dropped to the side when lifted.

The doctor continued to treat him for a week at home and then advised his removal to the Liverpool Children's Infirmary. He was admitted on March 10th, 1913.

On Admission.

The patient is an excessively healthy and well nourished boy, his cheeks are ruddy and complexion fair.

The skin is rather excessively moist. He is frightened and fretful and appears to suffer pain when moved in bed, notwithstanding this he is constantly crying for the nurse to put him on one or other side. He prefers this position to that of lying on his back.

The pain on pressure is most marked in the paralysed limbs. The head is well shaped, fontanelles closed.

Facial irritability is not increased. Ears and nose are well formed, no discharge from either.

Thorax and abdomen well formed. There are no enlarged glands to be felt in neck, axilla or groin and there is no evidence of previous rickets, syphilis or tuberculosis. The pulse rate is 128, respiration 24, temperature 100.2.

Abdominal palpation meets with voluntary resistance but reveals nothing abnormal.

Nervous System.

Cranium well formed, good shape. Circumference - 19¼". Fontanelles both closed. Vertebral column no rigidity or tenderness on pressure over spines.

There is no head retraction, but when the head is flexed forward the child cries with pain. Mental condition good. Sleeps fairly well.

Child is very irritable when disturbed. There is no delirium.

Motor Functions.

The muscular system appears to be well developed, but there is a marked flaccid paresis in both lower limbs. These are absolutely helpless and fall like a dead weight when lifted from the bed.

There is no marked difference in size between the two limbs.

Involuntary Movements.

There are no involuntary movements, spasms or convulsions.

Voluntary Movements.

Both lower limbs are absolutely helpless. Right arm normal. Left arm completely paralysed in following muscles - deltoid, biceps, triceps, brachial anticus.

Lower arm muscles - extensor, flexors and pronator and supinator, appear unaffected. Patient can grasp objects easily with left hand. He is unable to stand, to sit up or hold up his head, the back muscle groups appear quite paralysed.

No facial paralysis.

Reflexes.

Superficial. Abdominal cremasteris etc. are present but sluggish in response.

Deep. Complete loss of knee jerks and all other deep reflexes in lower limbs.

Organic. Child passes urine and faeces under him in bed, appears to have no control of sphincters.

Sensory Functions.

Appear unimpaired but owing to extreme fretfulness and tenderness it is very hard to estimate.

Trophic Symptoms.

Flabby muscles in upper left arm and both legs: no actual atrophy noted as yet.

Eyes.

Sight normal: pupils equal in size: shape normal, react to light and accomodation. No Nystagmus, squint or paralysis.

Ears.
Nothing. No discharge.

Digestive System.
Mouth dry. Tongue furred. Teeth good. Fauces clear. Appetite poor. No vomiting. Is very constipated.

Abdominal Examination:- reveals nothing, no lumps or fluid. Spleen and liver normal in size.

Respiratory System.
Thorax normal in shape. Bronchial sounds rather harsh, few dry rhonchi. Child has some bronchitis.

Circulatory System.
Heart - no enlargement or murmur.

Haemopoietic System.
Spleen is not enlarged. There are no palpable glands in neck, groin or axillae. Thymus and thyroid are normal.

Genito-urinary System.
No control over passage of urine. External genitalia - both testes down.
Urine - acid reaction. Specific gravity 1010. No abnormality present.

Skin.
No eruptions. Child perspires rather freely, especially at night.

Treatment - Urotropin V gr. T.D's.

Further Notes.
14.3.13. Child rather better. Tenderness not so marked

21.3.13. Massage begun.

27.3.13. Electrical treatment commenced. Will lie on back now for longer periods. Less tender. Appetite good. General condition better.
Paralysis just as complete as at first.

CASE V.

L.S. Female. Aged 2 yrs, 2 mos.

Admitted May 31st, 1912. Discharged June 26th, 1912. Result - Cured.

Complaint - weakness of left leg.

Family History.
Parents healthy except for rheumatism on mother's side. No miscarriages. Three other children alive and well.

Hygienic Surroundings at home - fair.

Previous Health.
Pneumonia twice as a baby. Measles and chickenpox.

Present Illness.
On May 22nd child was feverish and irritable. She vomited once and screamed when she was touched.
On May 24th she began to limp and lost the use of her left leg.
May 30th she seemed unable to sit up, the tenderness to touch had increased and she perspired freely. She complained of pain in her head, neck and back.

Admitted May 31st.
Condition then was as follows:-
An extremely well nourished and healthy little girl good colour, skin rather excessively moist: no eruptions etc. She is very irritable and screams when anyone even approaches her. Tenderness appears to be very acute especially in lower limbs. Head is well shaped, fontanelle closed. Facial irritability increased. Some enlargement of the cervical glands. Other glands normal. Abdomen - slightly distended probably gaseous, as child has been very constipated.
Left leg is quite helpless.

Nervous System.
The spine is slightly rigid. There is no head retraction but excessive pain is the result when any attempt is made to flex the head.
The child is extremely irritable and resents disturbance of any sort.

Motor Functions.
Complete paralysis of left leg, no movement is possible and on lifting the limb from the bed it drops back a dead weight.

Right leg is weaker than it normally should be.

Upper limbs are apparently unaffected. There is some weakness of the muscles of the back and the child is unable to hold up the head. There are no involuntary movements.

Electrical Reactions.

No reaction of degeneration present but reaction to galvanism is sluggish.

ACC > CCC

Faradic excitability - present.

Reflexes.

Superficial - present.

Deep - knee jerk left leg - lost. Right leg - normal.

Plantar reflex. Left - flexor responses sluggish.
Right - " " "

Organic reflex - no loss of control.

Trophic Functions.

There is marked wasting of the muscles of the left leg both in thigh and lower leg.

Measurements - Right calf - 7¼". Right thigh - 10"
Left calf - 7" Left thigh - 9⅜"

The Eyes are quite normal, pupils react to light and accomodation, no nystagmus or strabismus. Discs clear.

Ears - no pus or discharge.

Digestive System.

Tongue slightly furred, fauces somewhat reddened, no adenoid. Thirst is increased. Child has not vomited since admission.

Bowels very constipated.

Abdominal examination reveals nothing of note.

Rectal examination - nil.

Respiratory System. - nil.

Genito-urinary System.

No loss of sphincteric control.

Urine - specific gravity 1010, acid. No abnormal constituents.

Progress.

Urotropine was begun early and child seemed to respond very soon. The tenderness gradually passed off, the perspiration was not so profuse and the child became more friendly and began to take her food well.

The temperature varied greatly during the first week that she was in hospital, 97°-100°. It kept near the normal line however, after the first few days.

On June 9th massage was started and though the child did not take to this very kindly at first she gradually became more amenable.

June 25th, Improvement continues but owing to an outbreak of ileocolitis in the ward the child was sent home.

She attended the Out Patient Department twice weekly for massage and electrical treatment.

She was seen sometime later in January and February 1913, and was then walking quite well.

There is still some emaciation of the left leg especially of the thigh. She drags the left leg slightly and complains of a slight ache at times.

The knee jerk has returned but is sluggish.

Her bodily health is excellent and she can run about freely without support.

Massage is continued once weekly in order to try and get a still better result.

CASE VI.

J.H. Male. Aged 22 yrs.

Occupation - Insurance agent.

Admitted March 1st, 1913 to the Royal Infirmary, Liverpool. Discharged - May 8th 1913.

Result - much improved.

Complaint.

Patient is admitted complaining of inability to use his left arm, especially at the shoulder joint (duration 4 weeks).

Previous History.

There has been no hurt or injury to the arm in the past. Has good home in a healthy neighbourhood. He has never been a very robust man but has always enjoyed fairly good health. He is abstemious, takes no alcohol and smokes in moderation. For the last 6 months he has noticed that he has been rather nervous and despondent, and thinks that he has lost weight. He has never worked in lead.

Family History.

Single. Father died of pneumonia at age of 53. Mother always healthy. One brother and five sisters alive and well. No history of phthisis, syphilis or malignant disease.

Past Illnesses.

Measles, scarlatine in infancy. Has never had venereal disease. Suffers from neuralgia.

History of present Illness.

Four weeks ago (February 1st) patient went to bed, to all intents and purposes perfectly well. For a few days he had noticed some slight flabbiness of his left biceps but had taken no notice of this. The usual premonitory symptoms were entirely absent. The next morning (February 2nd) he awoke and found that he had pain in his left shoulder and upper arm, accompanied by loss of muscular power. He found great difficulty in dressing and especially in getting into his coat. These movements of dressing and undressing had been performed perfectly well the day before.

He went to see his doctor who said that he had developed paralysis and must go to an Infirmary. He was loth to do this and tried simple home remedies until the end of the month. Then as the loss of power seemed to be getting worse he took his doctor's advice and came to the Royal Infirmary, Liverpool, where he was admitted on March 1st to Ward XII.

Condition on Admission.

The patient can sit up in bed and has no pain whatsoever, not even on movement. He can take up any position he desires without inconvenience. There is no pyrexia and he seems quite well with the exception of his left arm. He weighs 8 st. 4lbs. and is 5ft.8" in height. He appears to have a somewhat neurotic temperament, is pale and thin but not emaciated.

Nervous System.

Patient is quite conscious and intelligent. He has no loss of smell, sight, taste, hearing or speech, no loss of optical accomodation and his pupils react to light normally.

Arms and Shoulders.

On looking at the patient from the front there is marked elevation of the left shoulder. The left trapezius and deltoid are soft and flabby. The supra spinatus, infraspinatus, and teres major muscles are also atrophied considerably and show a flaccid paralysis. Biceps and triceps are normal.

The left trapezius and deltoid are soft and flabby. The supraspinatus, infraspinatus and tenus major muscles are also atrophied considerably and show a flaccid paralysis. Biceps and triceps are weak.

Movements at Shoulder Joint.

Extension of the left upper arm cannot be performed His biceps retains some of its power however, and he can jerk his shoulder forwards by means of it. He also can perform a combined movement of extension and abduction by a forward movement of his scapula.

He cannot abduct, but retains the power of backward movement fairly well: his rhomboids are intact. Adduction is also possible.

On measurement of arms it is found that there is a difference of 1½" in girth of the two upper arms and 1½" in the forearms, the left being the smaller in each instance.

He seems to have excellent use of all his left forearm muscles. No numbness or tingling. There is, however, marked myotatic irritability of the scapular muscles.

Reflexes.

Superficial - exaggerated.

Deep - Triceps absent, supinator and wrist jerks - negative, knee jerk normal.

Organic - unimpaired.

Alimentary System.

Tongue slightly furred: very tremulous. Carious teeth. Is constipated.

Respiratory System.

Nothing to note.

Cardio-vascular System - nil.

Genito-urinary System - normal.

Treatment.

Tonics. Urotropin (grs X. T.D.'s) Massage and electricity, active and passive movements.

10.3.13. Condition slightly improved. Electrical reaction done. There is an incomplete reaction of degeneration in the affected muscles, the response to Faradism being much decreased, while the response to galvanism is almost lost.

12.3.13. The power in the biceps and deltoid appears to be somewhat improved. The deltoid is however, much weaker than normal.

24.3.13. Patient suffers a good deal from headache and is lethargic at times. Deltoid appears to be stronger.

May 8th. Much improved since admission. Can extend arm slightly at shoulder. Can abduct and adduct arm and exert a small amount of strength.
General condition much better.

Discharged.

CASE VII.

H.M. Male. Aged 8 years.

Admitted to the Children's Infirmary, Liverpool, Aug.7th 1912. Died Aug.7th, 1912.

Complaint. Paralysis of legs and left arm.

Family History.
Parents are both alive and well. There are eight other children all quite healthy.
Mother has had no miscarriages and no stillborn children.

Home surroundings - are good.

Previous Health.
The child has suffered from measles and bronchitis in infancy, but otherwise has been a healthy boy.

Present Illness.
On August 3rd, 1912, the patient was quite well. On August 4th he complained of abdominal pain, also of some soreness in the neck and legs. He was found to be unable to walk or move his arms.
The paralysis apparently came on quite suddenly. The parents can ascribe no cause for his illness and there appears to have been no noticeable prodromal symptoms. He gradually became worse up to the time of his admission to hospital.

On Admission.

The child is well nourished. Slightly flushed but shows no cyanosis. The muscular system is fairly well developed. The skin is extremely moist and perspiration is profuse. No eruption is present. He appears to be in pain but is able to lie flat on his back without trouble. Head is well shaped and shows no evidence of injury. Facial irritability is slightly increased. There are no enlarged glands to be palpated and no evidence of syphilis or tuberculosis can be found.

Respiration - 40. Temperature - 101F. Pulse - 120

Nervous System.

There is no head retraction, but on flexing the neck, pain is experienced by the boy and he cries out.

He is perfectly conscious and answers questions in a quick and intelligent manner. There is slight irritability but no delirium or affection of speech.

Motor Functions.

There is complete paralysis of both lower limbs from hip to foot; the left arm is also quite helpless, with the exception of the hand which can grasp weakly.

The right arm is not paralysed in its upper half, but the forearm is extremely weak and the grasp feeble. The elbow can be flexed and the shoulder shrugged slightly. Fingers can be moved.

The patient is able to sit up but does so with difficulty, he prefers to lie down. The neck can be moved but flexion causes pain.

Abdomen.

Only moves slightly in upper half on respiration. Chest does not move at all. Intercostals appear to be quite paralysed.

There are no involuntary movements, no tremors, spasms or convulsions.

Reflexes.

Superficial. Abdominal sluggish. Cremasteric - present.

Deep. Knee jerks absent. Triceps and biceps on left side negative; on right side present. Extensor supinator and forearm jerks are rather exaggerated in both arms. No ankle clonus is present. Babinski's sign is negative.

Organic. Patient has no control of sphincters and passes urine and faeces under him without appearing to notice it.

Sensory functions are unimpaired. Sensibility to touch, pain and hot and cold test tubes - normal. There is no wasting of muscles in any region. There is no great increase in tenderness to pressure.

Eyes.

Pupils small, react to light and accomodation. No nystagmus or squint.

Ears.

No discharge or alteration in hearing.

The tongue is dry and coated with thick white fur. There is some difficulty in swallowing.

Lumbar puncture.

Clear fluid - 40 c.c. withdrawn, slight excess pressure, no coagulation on standing. Albumen slightly in excess. Fehling reduced. Film shows excess of cells many lymphocytes and few polymorphs. No organisms seen.

Respiratory System.

Thorax is immobile on respiration. The extraordinary muscles of respiration are all working furiously, thyrohyoid, sternohyoid, etc.

Lungs.

Clear except for few moist rales at bases.

Circulatory System.

Heart is not enlarged. Apex beat in 4th intercostal space 2½" from midsternal line. There is a loud mitral systolic murmur heard at the apex.

Digestive System.

Tongue heavily coated and dry. Fauces slightly reddened. Extreme thirst. No vomiting. Bowels very constipated before admission, but since he came in large loose stools have been passed in bed. There is some difficulty experienced in deglutition.

Abdomen.

Flaccid, esaily palpable. The intestinal coils are easily seen. No lumps or tumour mass felt, no free fluid. Very little movement except in upper 1/3 where diaphragm is working.

Rectal examination - nothing.

Genito-urinary System.

No control over micturition. Urine slightly concen-

trated with water. No abnormal constituents.

Treatment. Progress. etc.

Urotropine (gr.V. every 2 hrs) was started as soon as possible after admission in the hope that some therapeutic result might occur, but it appeared to be useless.

The patient gradually became weaker and more drowsy. He could be roused from this lethargic condition but soon sank back again into his torpor.

He passed several involuntary motions under him and the urine trickled away. About 8 pm. he collapsed and his respiration became very weak, but with the help of stimulants he rallied and appeared to get rather better. He slept for a couple of hours and then gradually became worse, his respirations were short and rapid, there was extreme air hunger and some cyanosis and in the early morning he passed away, apparently from the increasing involvement of the respiratory centres. The temperature gradually rose till it reached 102°.

Post Mortem Examination.

Brain and spinal cord were removed and kept. (Special reference will be made to this later). The body generally was well nourished.

Heart - the mitral valve cusps were somewhat thickened but otherwise there was nothing to note.

Lungs - some congestion and oedema was present at the bases.

Abdomen - no enlargement of organs, viscera apparently healthy.

Kidneys - nothing.

The Brain was seen to be extremely congested on r emoving the skull cap: the meninges were oedematous and there was some adhesion of the lips of the Sylvian fissure. Otherwise nothing abnormal was discovered in the brain.

The Cord showed no macroscopic changes.

The cord was fixed and hardened and then sections were cut from the cervical dorsal and lumbar regions. In many nothing was noted, but in several sections, changes which appear to be confirmatory of the diagnosis of poliomyelitis were found. There were photographed and the prints are attached at the end of this work.

The most notable changes seem to be the round celled infiltration both perivascular and diffuse, this was noted in all regions but especially in the cervical and lumbar enlargements of the cord.

The anterior horn cells do not appear to have degenerated to any great extent but in some an unhealthy appearance exists with loss of nuclei.

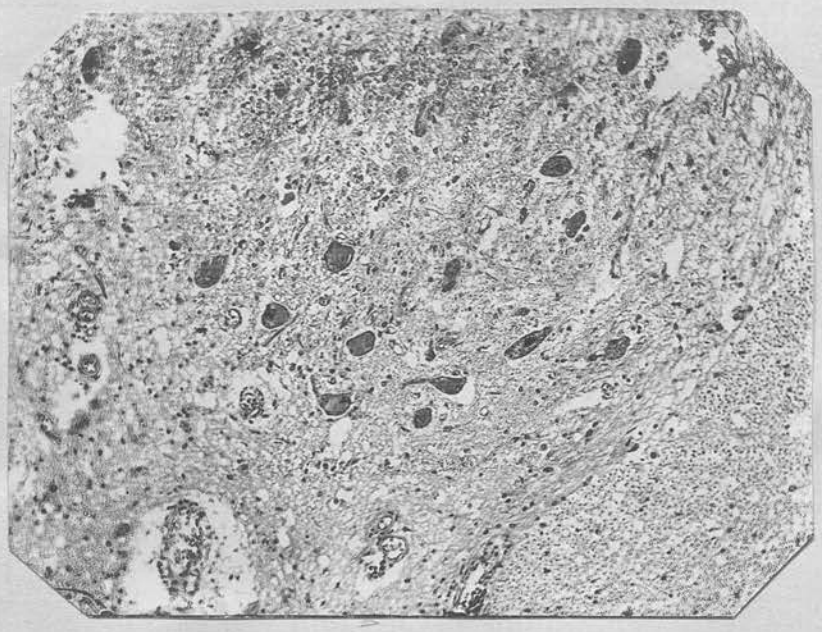
The sections were stained by a variety of methods. Haematoxylin, and eosin giving the best results. Marchis stain, Nissl's stain for cell granules and Van Giesen's process were also tried, but photographs of the best only are reproduced below.

CASE VI.

CASE VI.

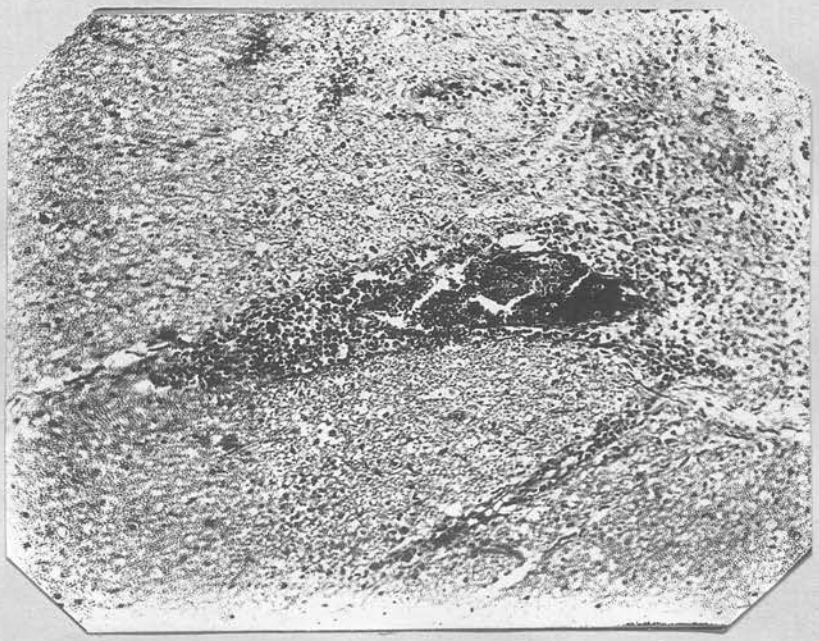
Case VII

MICROPHOTOGRAPHS FROM SECTIONS OF SPINAL CORD.



x 140. Stain - Haematin and Eosin.

No.I. Anterior horn cells from lumbar enlargement of cord. Several of the cells do not show a nucleus, others seem normal.

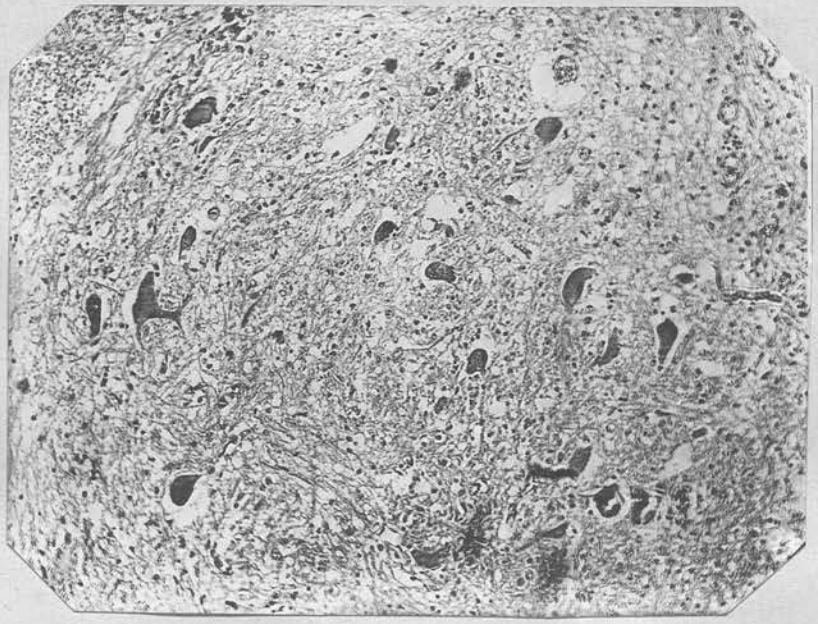


x 140. Stain - Haematin and Eosin.

No.II. Section of cervical enlargement showing intra and perivascular round celled infiltration.

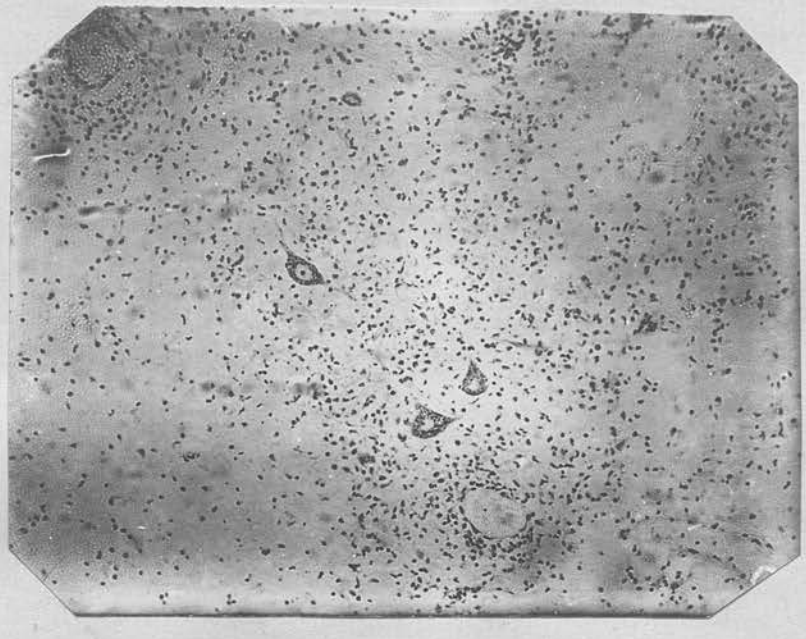
x 140. Stain - Haematin and Eosin.

No.III. Section from dorsal region.
Vessel showing round celled infiltration.



x 140. Stain - Van Giesen's.

No.IV. Lumbar enlargement. Cells of the anterior horn.
Some cells appear to be disintegrated having lost their nuclei and being of aberrant shape.

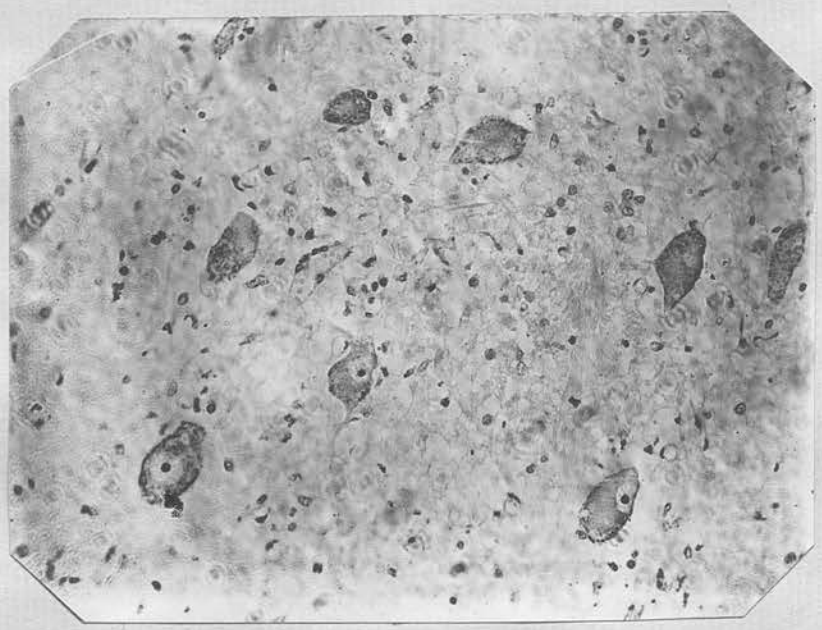


x 140 Stain - Nissl's.

No. V. Section from cervical region.
Cells show Nissl granules plainly, these do not seem as yet to be affected. Diffuse round celled infiltration is marked.

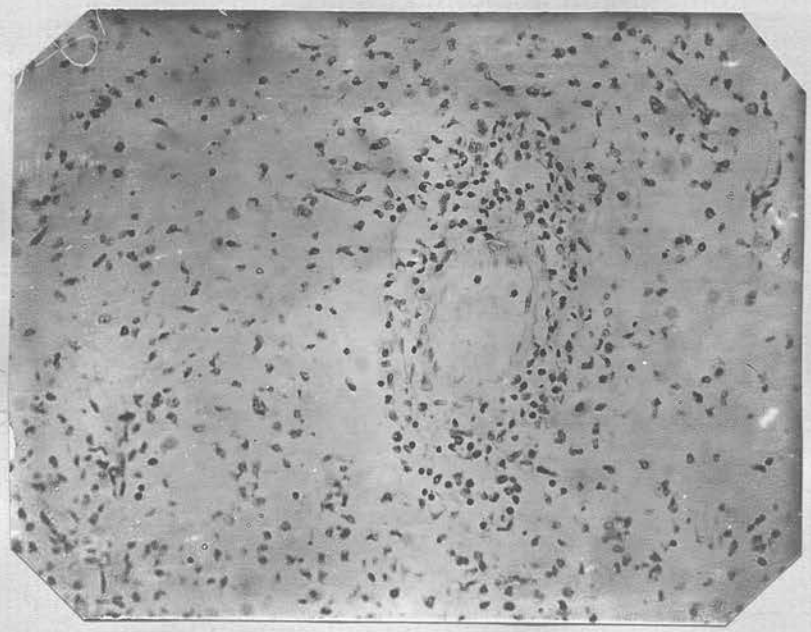
x 140. Stain - Haematin and Eosin.

No.VI. Section of lumbar enlargement.
Shows well the interstitial meningitis in the anterior fissure of the cord.



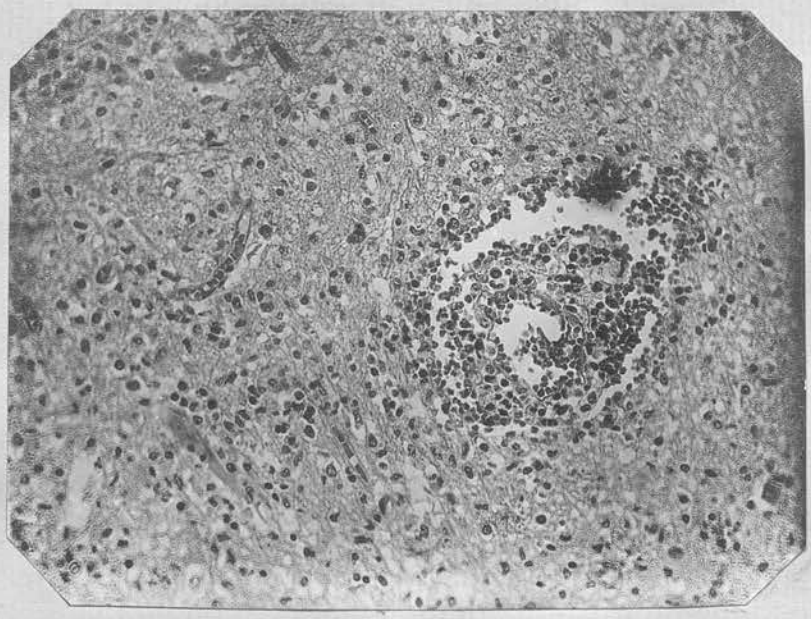
x 250. Stain - Nissl's.

No.VII. Section of lumbar enlargement. Higher power. Anterior horn cells are seen. Nissl granules are indistinct in several cells.



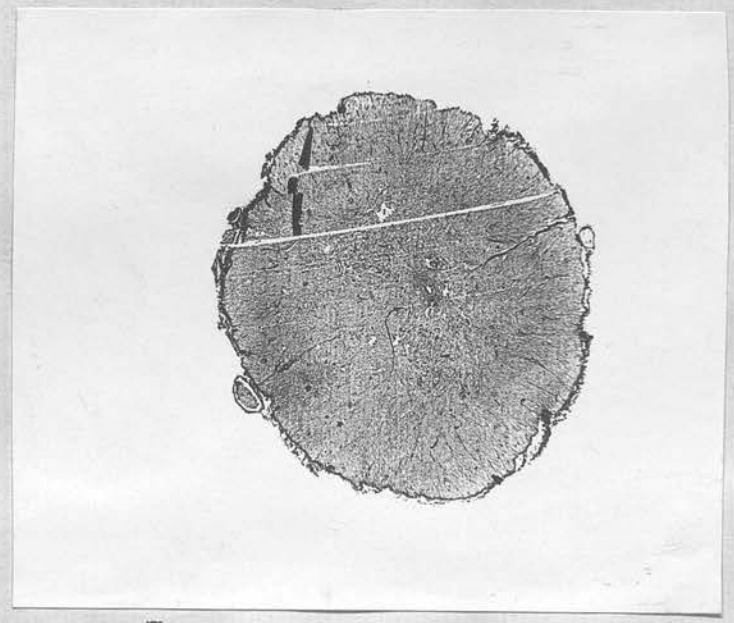
x 250. Stain - Nissl's.

No.VIII. Section from cervical region. Well marked preivascular round celled infiltration with diffuse lymphocytic infiltration of gray matter.



x 250. Stain - Haematin and Eosin.

No.IX. Section from cervical region.
Vessel shewing excessive perivascular infiltration and also great number of round cells in surrounding area.



x 7

No.X. Whole section. From lumbar enlargement.
Gray matter can be faintly made out with anterior horn nerve cells.